

THE BRITISH
JOURNAL OF SURGERY

THE BRITISH JOURNAL OF SURGERY

UNDER THE DIRECTION OF THE FOLLOWING
EDITORIAL COMMITTEE :

LORD MOYNIHAN, K.C.M.G., C.B. (Leeds), *Chairman*

- | | |
|---|---|
| H. T. DYKE ACLAND, C.M.G., C.B.E. (Christchurch, N.Z.) | ROBERT E. KELLY, C.B. (Liverpool) |
| JOHN ANDERSON, D.S.O. (Dundee) | J. R. LEARMONTH (Aberdeen) |
| EDWARD ARCHIBALD (Montreal) | HUGH LETT, C.B.E. (London) |
| SIR C. ARTHUR BALL, Bart. (Dublin) | ADAMS A. McCONNELL (Dublin) |
| SEYMOUR BARLING, C.M.G. (Birmingham) | B. C. MAYBURY (London) |
| WILLIAM H. BOWEN (Cambridge) | WM. ERNEST MILES (London) |
| L. R. BRAITHWAITE (Leeds) | SIR HENRY S. NEWLAND, C.B.E., D.S.O. (Adelaide) |
| ARTHUR H. BURGESS (Manchester) | CHARLES A. PANNETT (London) |
| SIR F. POWELL CONNOR, D.S.O., Lt.-Col. I.M.S. (Calcutta) | HARRY PLATT (Manchester) |
| H. B. DEVINE (Melbourne) | SIR D'ARCY POWER, K.B.E. (London) |
| HAROLD R. DEW (Sydney) | ALFRED W. SHEEN, C.B.E. (Cardiff) |
| GEORGE H. EDINGTON (Glasgow) | GRAHAM S. SIMPSON (Sheffield) |
| R. C. ELMSLIE, O.B.E. (London) | WILFRED TROTTER (London) |
| CHARLES H. FAGGE (London) | G. GREY TURNER (Newcastle-on-Tyne) |
| C. H. S. FRANKAU, C.B.E., D.S.O. (London) | CECIL P. G. WAKELEY (London) |
| JOHN FRASER, M.C. (Edinburgh) | MAJ.-GEN. J. W. WEST, C.M.G., C.B.E. (London) |
| ANDREW FULLERTON, C.B., C.M.G. (Belfast) | SIR W. I. de COURCY WHEELER (London) |
| GEORGE E. GASK, C.M.G., D.S.O. (London) | D. P. D. WILKIE, O.B.E. (Edinburgh) |
| W. SAMPSON HANDLEY (London) | DAVID STORER WYLIE, C.M.G., C.B.E. (Palmerston North, N.Z.) |
| GEOFFREY JEFFERSON (Manchester) | ARCHIBALD YOUNG (Glasgow) |
| E. K. MARTIN (London), <i>Editor, Atlas of Pathological Anatomy</i> | |
| E. W. HEY GROVES (Bristol), <i>Editorial Secretary</i> | |

VOLUME XXI.

July 1933 to April 1934. Numbers 81 to 84.

Together with the Atlas of Pathological Anatomy, Numbers 33 to 36.

BRISTOL: JOHN WRIGHT & SONS LTD.

LONDON: STATIONERS' HALL COURT. SIMPKIN MARSHALL LTD

TORONTO: THE MACMILLAN CO. OF CANADA LTD.

CALCUTTA: BUTTERWORTH AND CO. (INDIA) LTD.; THACKER, SPINK AND CO. BOMBAY: W. THACKER AND CO.
SYDNEY: ANGUS AND ROBERTSON LTD. NEW ZEALAND: WHITCOMBE AND TOMBS LTD.

UNITED STATES OF AMERICA: WILLIAM WOOD AND CO.. BALTIMORE, *Sole Agents.*

PRINTED IN GREAT BRITAIN
BY JOHN WRIGHT AND SONS LTD., BRISTOL

CONTENTS OF VOLUME XXI

NUMBER 81.

						PAGE
SOME EARLY SURGICAL CASES :—						
The Edwin Smith Papyrus	-	-	-	-	<i>Sir D'Arcy Power</i>	1
SYMPATHECTOMY AS AN EXPERIMENT IN HUMAN PHYSIOLOGY					<i>J. Paterson Ross</i>	5
A CASE OF PARATHYROID TUMOUR	-	-	<i>Geoffrey Keynes and Hermon Taylor</i>			20
POST-TRAUMATIC EPIDERMOID CYSTS OF HANDS AND FINGERS					- <i>E. S. J. King</i>	29
AN INVESTIGATION OF 742 CASES OF HEMATURIA					- <i>R. K. Debenham</i>	44
A CASE OF RENAL AND URETERIC CALCULI OF UNUSUAL SIZE. REPEATED FORMATION OF CALCULI					- <i>Edgar Freshman</i>	63
OSTEO-CHONDRITIS DISSECANS					- <i>H. A. T. Fairbank</i>	67
THE ANATOMY OF THE PERIPHERAL SYMPATHETIC NERVOUS SYSTEM						
					<i>H. H. Woollurd and R. E. Norrish</i>	83
MESENTERIC CYST AS A CAUSE OF INTESTINAL OBSTRUCTION					<i>W. Arklay Steel</i>	104
A CASE OF VESICAL PAPILLOMA WITH WIDESPREAD METASTASES					<i>M. F. Nicholls</i>	108
THE SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM					- <i>G. E. Gask</i>	113
PIED FORCÉ OR MARCH FOOT					- <i>Harold Dodd</i>	131
SHORT NOTES OF RARE OR OBSCURE CASES					- <i>Various Writers</i>	145
REVIEWS AND NOTICES OF BOOKS					- - - - -	161

NUMBER 82.

THE CLINICAL ASPECTS OF BRANCHIAL FISTULE	-	-	-	<i>Hamilton Bailey</i>	173
THE DIVERTICULA OF THE JEJUNO-ILEUM	-	-	-	- <i>Ian Fraser</i>	183
A CASE OF LIPOMA OF THE MESENTERY	-	-	-	<i>Frank d'Abreu</i>	212

	PAGE
OCCUPATIONAL ANEURYSM OF THE PALMAR ARTERIES - <i>D. Stewart Middleton</i>	215
COCYGEAL SINUS - - - - - <i>R. L. Newell</i>	219
ASSOCIATED FACIAL AND INTRACRANIAL HÆMANGIOMATA - <i>Lambert Rogers</i>	229
POUCHES OF THE PHARYNX AND ŒSOPHAGUS WITH SPECIAL REFERENCE TO THE EMBRYOLOGICAL AND MORPHOLOGICAL ASPECTS - - - <i>R. W. Raven</i>	235
THE SURGICAL TREATMENT OF BRONCHIECTASIS - - - <i>Robert M. Jones</i>	257
PULMONARY LOBECTOMY. TECHNIQUE AND REPORT OF TEN CASES <i>J. E. H. Roberts and H. P. Nelson</i>	277
BRONCHIECTASIS : A STUDY OF THE PATHOLOGY OF SIXTEEN SURGICAL LOBECTOMIES FOR BRONCHIECTASIS - - - - - <i>W. L. Robinson</i>	302
ETIOLOGY AND TREATMENT OF SLIPPED EPIPHYSIS OF THE HEAD OF THE FEMUR <i>E. N. Wardle</i>	313
OBSERVATIONS UPON MULTIPLE INTRAMESENTERIC DIVERTICULA OF THE SMALL INTESTINE - - - - - <i>R. Weedon Butler</i>	329
CLEFT PALATE - - - - - <i>W. E. M. Wardill</i>	347
SHORT NOTES OF RARE OR OBSCURE CASES - - - - - <i>Various Writers</i>	370
REVIEWS AND NOTICES OF BOOKS - - - - -	376

NUMBER 83.

IN MEMORIAM :—

Sir George Minkins, G.C.M.G., C.B., LL.D. - - - - -	381
PRESENTATION TO THE EDITORIAL SECRETARY - - - - -	383
SOME EARLY SURGICAL CASES :—	
The Edwin Smith Papyrus - - - - - <i>Sir D'Arcy Power</i>	385
CARCINOMA OF THE KIDNEY - - - - - <i>Graham Simpson</i>	388
RIGHT PARADUODENAL HERNIA - - - - - <i>A. C. Halliwell</i>	398
TWO CASES OF OSSIFICATION IN THE INTERNAL SEMILUNAR CARTILAGE <i>H. Jackson Burrows</i>	404
THE FALLACY OF EXPECTANT TREATMENT IN ACUTE APPENDICITIS <i>H. C. W. Nuttall</i>	411

CONTENTS

vii

	PAGE
THE PATHOLOGY, DIAGNOSIS, AND TREATMENT OF CONGENITAL DIAPHRAGMATIC HERNIA IN INFANTS - - - <i>N. R. Barrett and C. E. W. Wheaton</i>	420
PROSTATECTOMY WITH CLOSURE: FIVE YEARS' EXPERIENCE <i>S. Harry Harris</i>	434
ENLARGEMENT OF THE ANTERIOR PORTION OF THE PROSTATE GLAND <i>B. S. Cran</i>	453
AN INQUIRY INTO THE RESULTS OF SURGICAL TREATMENT OF GENITAL TUBERCULOSIS IN THE MALE - - - - <i>Rolph O. Lee and Kenneth Bowes</i>	456
CALCIFICATION, DECALCIFICATION, AND OSSIFICATION <i>R. W. Jones and R. E. Roberts</i>	461
A CASE OF HYPERPARATHYROIDISM WITH CERTAIN UNUSUAL FEATURES <i>T. W. Minpriss and R. W. Butler</i>	500
THE OCCURRENCE OF SCROTAL HERNIA IN MICE UNDER TREATMENT WITH OESTRIN <i>Harold Burrows</i>	507
BENIGN GIANT-CELL TUMOUR OF THIRD CERVICAL VERTEBRA: A CASE REPORT <i>J. A. Macfarlane and Eric A. Linell</i>	513
ADENOPAPILLOMA OF THE STOMACH ASSOCIATED WITH LYMPHOID HYPERPLASIA OF THE DUODENUM - - - - <i>A. E. Webb-Johnson and E. G. Muir</i>	519
SPONTANEOUS DISLOCATION OF THE HIP IN CHILDHOOD - - - <i>P. N. Ray</i>	523
TWO CASES OF KILLOID FORMATION, WITH COMMENTS - - <i>C. E. L. Burman</i>	527
ACUTE INFECTIVE OSTEOMYELITIS OF THE TIBIA: USE OF WINNETT ORR TECHNIQUE <i>M. G. O'Malley</i>	530
SHORT NOTES OF RARE OR OBSCURE CASES - - - <i>Various Writers</i>	532
REVIEWS AND NOTICES OF BOOKS - - - - - - - -	547

NUMBER 84.

IPSISSIMA VERBA:—

Lister and Wiring the Patella - - - - <i>Sir D'Arey Power</i>	557
A CONTRIBUTION TO THE STUDY OF PULMONARY LOBECTOMY <i>A. Graham Bryce</i>	560
MULTIPLE CARCINOMA OF THE COLON: WITH FOUR ORIGINAL CASES <i>A. J. Cokkinis</i>	570
SOME EXPERIMENTAL OBSERVATIONS BEARING ON THE ETIOLOGY OF MEGACOLON <i>Harold Burrows</i>	577
THE PATHOLOGY OF ACUTE STRANGULATION OF THE INTESTINE <i>Robert L. Holt</i>	582

	PAGE
NEUROSURGERY IN THE TREATMENT OF DISEASES OF THE PERIPHERAL BLOOD-VESSELS <i>D. J. MacMyn</i>	604
THE SELENIDE TREATMENT OF CANCER - - - - <i>A. T. Todd</i>	619
BILHARZIA DISEASE IN ENGLAND - <i>J. B. Christopherson and R. Ogier Ward</i>	632
SPINDLE-CELLED MESENTERIC TUMOURS : WITH REMARKS ON SIMILAR RETRO- PERITONEAL TUMOURS - - - - <i>H. A. Phillips</i>	637
OBSERVATIONS ON PLEURAL ABSORPTION - - - <i>R. C. Brock</i>	650
THE PLAN OF THE VISCERAL NERVES IN THE LUMBAR AND SACRAL OUTFLOWS OF THE AUTONOMIC NERVOUS SYSTEM - - - - <i>Hugh C. Trumble</i>	664
MALIGNANT ADENOMA OF THE PROSTATE WITH SECONDARY GROWTHS IN THE VERTE- BRAL COLUMN SIMULATING POTT'S DISEASE <i>W. E. Carnegie Dickson and T. R. Hill</i>	677
PRIMARY CARCINOMA OF THE LIVER : WITH REPORT OF A CASE SUCCESSFULLY TREATED BY PARTIAL HEPATECTOMY - - - - <i>A. Lawrence Abel</i>	684
SHORT NOTES OF RARE OR OBSCURE CASES - - - <i>Various Writers</i>	701
REVIEWS AND NOTICES OF BOOKS - - - - - - -	709

GENERAL INDEX TO VOLUME XXI.

SUPPLEMENT: ATLAS OF PATHOLOGICAL ANATOMY.
FASCICULUS IX: DISEASES OF THE GENITO-URINARY SYSTEM AND OF
THE APPENDIX.

THE BRITISH JOURNAL OF SURGERY

VOL. XXI.

JULY, 1933.

No. 81.

SOME EARLY SURGICAL CASES.

By SIR D'ARCY POWER, K.B.E., LONDON.

I. THE EDWIN SMITH PAPYRUS.

THE story of the Edwin Smith Papyrus has already been told in the pages of the JOURNAL (1930, xviii, 341, and 1932, xx, 34), but the cases are so interesting that they are worthy of a more careful study from the purely surgical side. The original writer of the papyrus was a surgeon living in Egypt at a time when the Pyramids were being built, probably between 3000 and 2500 B.C. The original work was re-edited in the seventeenth century B.C. by a surgeon who found that many of the terms used had become obsolete. These old terms he interpreted by a gloss and added a commentary explaining what he thought his author had meant to say. The original writer and his editor had the same type of mind as a modern surgeon. They were concerned with facts alone and had no use for magic, charms, or more than the simplest medicines.

The papyrus is a mere fragment, wanting at the beginning and ending abruptly in the middle of a sentence. There is sufficient to show that it was a systematic text-book of surgery dealing with wounds and external injuries, starting with the head and proceeding downwards to the face, neck, and chest. The writer follows a regular plan—first a statement of the injury, then the results of the examination followed by a diagnosis and the appropriate treatment. Some of the injuries he can cure, others he says that he will try to cure, others again he recognizes as fatal, and with these he will have nothing to do. In a true scientific spirit he records the signs and the diagnosis of the injuries for which he considered there was no cure. Care must be taken not to read our own knowledge into the facts, but it seems that he knew something about the action of the heart, something about the brain, and something about the spinal cord. He stitched incised wounds and recognized that the stitches became loose when the wound was healed. He applied a first dressing of fresh meat and afterwards a dressing of honey made up into an ointment. Adhesive plasters he used extensively. Bandaging was a fine art, as may be seen in the wrapping of mummies, and splints he used according to methods which had already become standardized. All the forty-eight cases

are interesting, and the following are selected to illustrate his methods and ideas. It may be noted that the patients were treated sitting up, and not, as is now the custom, in a recumbent posture.

If thou examinest a man with a gaping wound in his head penetrating to the bone and perforating the skull; thou shouldest palpate his wound; shouldest thou find him unable to look at his two shoulders and his breast and suffering with stiffness in his neck. Thou shouldest say regarding him "One having a gaping wound in his head penetrating to the bone and perforating his skull while he suffers with stiffness in his neck. An ailment which I will treat."

Now after thou hast stitheed it thou shouldest lay fresh meat upon his wound the first day. Thou shouldest not bind it. Moor him at his mooring stakes until the period of his injury passes by. Thou shouldest treat it afterward with grease, honey and lint every day until he recovers.

To this account in the original text the surgeon who edited the work some seven or eight hundred years later adds four explanations or glosses. He says: (1) "Perforating his skull." It means his skull, a contracted smash, through his incurring a break like a puncture of a jar. (2) "Unable to look at his two shoulders and his breast." It means it is not easy for him to look at his two shoulders and it is not easy for him to look at his breast. (3) As for "stiffness of the neck", it means a lifting up from his having incurred this injury, which has shifted into his neck so that his neck also suffers with it. (4) "Moor him at his mooring stakes." It means putting him on his customary diet without administering to him a prescription. In the light of the context the original writer and his editor seem to mean by this unusual expression "Do nothing, keep him at rest."

There are two cases of fractured base. The first he will try to cure, the second, associated with a depressed fracture, he will not undertake.

Instructions concerning a gaping wound in his head penetrating to the bone and splitting his skull:—

If thou examinest a man having a gaping wound in his head, penetrating to the bone and splitting his skull thou shouldest palpate his wound. Shouldest thou find something disturbing therein under thy fingers and he shudders exceedingly whilst the swelling which is over it protrudes, he discharges blood from both nostrils and from both his ears, he suffers with stiffness in his neck so that he is unable to look at his two shoulders and his breast. Thou shouldest say regarding him; "One having a gaping wound in his head penetrating to the bone and splitting his skull; while he discharges blood from both his nostrils and from both his ears and he suffers with stiffness in his neck. An ailment with which I will contend."

Now when thou findest that the skull of the man is split thou shouldest not bind him but moor him at his mooring stakes until the period of his injury passes by. His treatment is sitting. Make for him two supports of brick until thou knowest that he has reached a decisive point. Thou shouldest apply grease to his head and soften his neck therewith and both his shoulders. Thou shouldest do likewise for every man whom thou findest having a split skull.

The gloss added by the editor explains: (1) "Splitting his skull" means separating shell from shell of his skull while fragments remain sticking in the flesh of his head and do not come away. (2) "The swelling which is over it protrudes" means that the swelling which is over this split is large, rising upward. (3) As for "until thou knowest he has reached a decisive point", it means until thou knowest whether he will live or die he is a case of an "ailment with which I will contend."

A gaping wound of the head with compound comminuted fracture of the skull.

Instructions concerning a gaping wound in his head penetrating to the bone smashing the skull and rending open the brain of his skull:—

If thou examinest a man having a gaping wound in his head penetrating to the bone, and smashing his skull: thou shouldest palpate his wound. Shouldest thou find that smash which is in his skull deep and sunken under thy fingers whilst the swelling which is over it protrudes, he discharges blood from both his nostrils and both his ears and he suffers with stiffness in his neck so that he is unable to look at his two shoulders and his breast. Thou shouldest say regarding him “One having a gaping wound in his head penetrating to the bone and smashing his skull while he suffers with stiffness in his neck. An ailment not to be treated.”

Thou shalt not bind him, but moor him to his mooring stakes until the period of his injury passes by.

The gloss adds: As for “smashing his skull”, it means a smash of his skull such that bones getting into that smash sink into the interior of his skull.

Perhaps the earliest reference to the brain is the following:—

If thou examinest a man having a gaping wound in his head penetrating to the bone, smashing his skull and rending open the brain of his skull thou shouldest palpate the wound. Shouldest thou find that smash which is in his skull like those corrugations which form in molten copper and something therein throbbing and fluttering under thy finger like the weak place of an infant's crown before it becomes whole—when it has happened there is no throbbing and fluttering under thy fingers until the brain of his skull is rent open and he discharges blood from both his nostrils and he suffers from stiffness in his neck. Thou shouldest say “An ailment not to be treated.” Thou shouldest anoint that wound with grease. Thou shalt not bind it; thou shalt not apply two strips upon it; until thou knowest that he has reached a decisive point.

The glosses are somewhat more illuminating than usual: (1) As for “smashing his skull and rending open the brain of his skull”, it means the smash is a large opening to the interior of the skull, to the membrane enveloping the brain so that it breaks open the fluid in the interior of his head. (2) As for “those corrugations which form on molten copper”, it means copper which the coppersmith pours off before it is forced into the mould because of something foreign upon it like wrinkles. It is like ripples of pus.

It is clear from this case that the original surgeon had knowledge of the cerebral membranes and of the cerebrospinal fluid. He had seen the convolutions of the brain during life, perhaps as the result of a wound in battle with a heavy mace.

The next case appears to be one of septic cerebral thrombosis following an injury. It begins:—

If thou examinest a man having a gaping wound in his head, penetrating to the bone and perforating the sutures of his skull thou shouldest palpate his wound although he shudders exceedingly. Thou shouldest cause him to lift his face; if it is painful for him to open his mouth and his heart is too weary to speak [beats feebly (?)]; if thou observest his spittle hanging at his two lips and not falling off, whilst he discharges blood from both his nostrils and from both his ears; he suffers with stiffness in his neck and is unable to look at his two shoulders and his breast. Thou shouldest say concerning him “One having a gaping wound in his head penetrating to the bone and perforating the sutures of his skull; the cord of his mandible is contracted; he discharges blood from both his nostrils and from both

his ears while he suffers with stiffness of his neck. An ailment with which I will contend." Now as soon as thou findest that the cord of that man's mandible, his jaw, is contracted thou shouldest have made for him something hot until he is comfortable so that his mouth opens. Thou shouldest bind it with grease, honey and lint until thou knowest that he has reached a decisive point—(that is to say whether he is going to get well or die).

The surgeon then proceeds to give the symptoms which he considers foretell a fatal result :—

If thou findest that the flesh of that man has developed fever from that wound which is in the sutures of his skull, while that man has developed *ty* (meaning of the word unknown) from that wound thou shouldest lay thy hand upon him. Shouldest thou find his countenance is clammy with sweat, the ligaments of his neck are tense, his face is ruddy, his teeth and his back [?] the odour of the chest of his head is like the urine of sheep, his mouth is bound and both his eyebrows are drawn, whilst his face is as if he wept. Thou shouldest say regarding him "One having a gaping wound in his head penetrating to the bone, perforating the sutures of his skull; he has developed *ty*, his mouth is bound and he suffers with stiffness in his neck. An ailment not to be treated."

If however thou findest that that man has become pale and has already shown exhaustion. Thou shouldest have made for him a wooden brace padded with linen and put into his mouth. His treatment is sitting placed between two supports of brick until thou knowest that he has reached a decisive point.

The next case is also of great interest. It shows that the surgeon had observed the paralysis attending an injury to the brain. The translation reads :—

Instructions concerning a smash in his skull under the skin of his head.

If thou examinest a man having a smash of his skull under the skin of his head, while there is nothing at all upon it, thou shouldst palpate his wound. Shouldest thou find that there is a swelling protruding on the outside of that smash which is in his skull, while his eye is askew because of it, on the side of him having that injury which is in his skull; and he walks shuffling with his sole on the side of him having that injury which is in his skull.

Thou shouldest account him one whom something entering from outside has smitten, as one who does not release the head of his shoulder fork and one who does not fall with his nails in the middle of his palm; whilst he discharges blood from both his nostrils and from both his ears and he suffers from stiffness in his neck. An ailment not to be treated.

The treatment is sitting until thou knowest he has reached the decisive point.

The glosses explain : (1) "He walks shuffling with his sole." The surgeon means walking with his sole dragging so that it is not easy for him to walk when it (the sole) is feeble and turned over while the tips of his toes are contracted to the ball of his sole and they (the toes) walk fumbling the ground. (2) "Something entering from the outside." It means the breath of an outside god or death, not the intrusion of something which his flesh engenders. Both the original writer and his editor are trying to distinguish between the results of an injury and of such internal causes as apoplexy or epilepsy which were associated in their minds with demoniac possession.

(To be continued.)

SYMPATHECTOMY AS AN EXPERIMENT IN HUMAN PHYSIOLOGY.*

By J. PATERSON ROSS,

FROM THE SURGICAL PROFESSORIAL UNIT, ST. BARTHOLOMEW'S HOSPITAL.

OUR understanding of the functions of the human sympathetic nervous system is very imperfect. for clinical experience not only reveals gaps in our knowledge, but also casts grave doubt upon many current beliefs. Though it would be deplorable to detract one whit from the honour due to the great physiologists of the past for their early investigations, it is important to realize that the accepted ideas with regard to the workings of the sympathetic system in man have been derived from experiments upon the lower animals, whose nervous system in general, and whose sympathetic system in particular, differ in many respects from the human. It is only since surgery, the most fruitful method of research into the problems of human physiology and pathology, has embraced the sympathetic system, that we have begun to understand something of its activity in man. A great deal has been disclosed already, but surgical experiments take months or even years to yield their results, and it must be remembered that a large number of our present impressions are founded upon incomplete experiments, and the impressions may require considerable revision and modification before they develop into conclusive evidence. Our debt to the physiologists lies in the encouragement and the direction which their animal experiments have given us in recognizing general principles, in devising operative procedures, and in showing how accurate observations may be recorded.

The collection and careful analysis of information obtained by accurate methods of observation of patients, and more especially of patients before and after an operation has been performed upon some portion of the sympathetic system, will bring to light facts which can be proved by no other method of research, and will be the foundation for hypotheses to be tested by further surgical investigation.

Already sympathectomy has been performed for a very large number of diverse conditions, and it would be tedious and unprofitable to summarize what is known about the whole system. I propose to limit myself mainly to the consideration of the value of the operation of sympathectomy in elucidating the physiology of vasomotor control, of intestinal movements, and of micturition, and as a contribution to our knowledge of sensory pathways in the sympathetic system.

VASOMOTOR CONTROL.

Let us consider first the path for vasoconstrictor impulses to the upper extremity. The connector cells lie in the lateral horn of the grey matter of

* A Hunterian Lecture delivered at the Royal College of Surgeons, February 3, 1933.

the thoracic portion of the spinal cord, from as high as the fourth to as low as the ninth segment. Preganglionic fibres pass from these cells by the white rami communicantes to the ganglionated trunk of the sympathetic, in which they ascend to terminate around the ganglion cells of the middle and inferior cervical and the first and second thoracic ganglia. The inferior cervical and first thoracic ganglia are commonly fused and form the stellate ganglion. In these ganglia lie the excitor cells, whose axons, the postganglionic fibres, run in the grey rami to join the roots of the brachial plexus.

Section of all the grey rami passing to the brachial plexus is a surgical impossibility, and section of the thoracic sympathetic trunk below the second ganglion is unsatisfactory because regeneration can take place, and also because it is probably true to say that peripheral vasodilatation is maximal only if the cells from which the postganglionic fibres originate are removed. The only satisfactory method, therefore, of removing the sympathetic supply to the arm is by ganglionectomy, and the results of the many operations which have now been carried out show that the section of the visceral branches of the ganglia has no appreciable ill effect upon the patient.

The sympathetic supply to the legs has its connector cells in the tenth, eleventh, and twelfth thoracic and the first and second lumbar segments, the preganglionic fibres ending in the lumbar ganglia below the second and in the sacral ganglia. The grey rami from the second lumbar ganglion and the ganglia below it contain postganglionic fibres which are distributed to the periphery by way of the lumbar and sacral plexuses of the spinal nerves. It is therefore clear that excision of the second, third, and fourth lumbar ganglia and the intervening portions of the trunk will mean removal of the excitor cells for the lumbar nerves and division of all the preganglionic fibres to the sacral nerves. In practice this operation seems to provide an adequate degree of vasodilatation even in the foot, though it is probable that to produce full dilatation it would be necessary to remove the sacral ganglia as well, a step which would add very considerably to the difficulty and risk of the operation. It is therefore fortunate that ablation of the sacral ganglia seems to be unnecessary.

Removal of the second, third, and fourth lumbar ganglia involves the section of certain important visceral branches passing from these ganglia to join the aortic, inferior mesenteric, and superior hypogastric plexuses. On this consideration ramisectomy was practised for some time; but as the section of these visceral branches is not a disadvantage to the patient, and may, in certain circumstances, be beneficial, ganglionectomy is now preferred to ramisectomy.

METHODS OF INVESTIGATION.

Sympathetic nerve-fibres have been traced to the minute terminal branches of the vascular system, but the most striking effect of sympathectomy is dilatation of the arteries, and the simplest method of estimating the resultant increase in blood-flow is to record the rise of temperature in the limb. The most accurate temperature records for clinical purposes may be obtained by using copper-constantan thermocouples, which are attached to the skin by adhesive strapping. So that readings may be taken from several

SYMPATHECTOMY AND HUMAN PHYSIOLOGY 7

parts simultaneously, the wires from the couples pass through a simple key to the circuit, which includes a mirror galvanometer and a vessel of known constant temperature.

Before an operation for sympathectomy is undertaken it is important to be able to foretell the amount of benefit which is to be expected from it, and various methods of inhibiting or paralysing sympathetic nerves are employed in order to make such a forecast. The peripheral vasodilatation associated with a rise in body temperature is due to inhibition of sympathetic vasoconstrictor impulses, and for experimental purposes the body temperature may be raised either by protein shock or by heating the body in a hot-air bath.

Brown has described the reliance he has come to place upon the first method after several years' experience, more particularly in differentiating organic obstruction of arteries from conditions in which the lumen is narrowed by spasm. He induces fever by intravenous injection of triple typhoid vaccine, and the surface temperature of the fingers and toes is taken simultaneously with the mouth temperature. Normally, after a slight fall, the temperature in the mouth and on the surface rises, the magnitude of the surface rise depending on the initial temperature of the extremity, the severity of the febrile reaction, and the patency of the arteries. If the rise of temperature in the mouth is subtracted from the rise of skin temperature, the figure so obtained will indicate the change in skin temperature due to movement of blood into the extremity as a result of vasomotor changes.

What Brown calls the 'vasomotor index' is determined by dividing the above figure by the rise of temperature in the mouth, i.e.,

$$\frac{\text{surface rise} - \text{mouth rise}}{\text{mouth rise}}$$

and this index is high in cases of vasomotor disease associated with spasm, while in conditions of organic disease in which vasodilatation is impossible the value drops to zero.

In our hands, however, the alternative of warming the body in a hot-air bath, following the plan suggested by Lewis,¹ has given us even more information, we imagine, than could be obtained by protein shock, since the experimental conditions are more directly under our control, and small changes in the behaviour of different digits during vasodilatation may be more accurately observed.

The patient sits in a small chamber made of draught-proof non-conducting material, so arranged that with the coverings open the temperature within may remain about the same as that of the room; but when the chamber is closed, the coverings being made to fit closely at the neck and wrists (or ankles), the temperature within it may be raised rapidly to 50° C. by means of a series of carbon-filament lamps. The hands, previously cooled by immersion for twenty minutes in a water bath at 15° C., protrude from orifices in the coverings and rest upon a small raised platform of wood or cork so as not to come into contact with the chamber itself. The room temperature should be kept low—about 15° C. is probably the optimum—for at higher temperatures vasodilatation might be produced by external warmth, whereas what we wish to determine is the degree of vasodilatation produced

reflexly owing to heating the body. It is an easy matter to raise the room temperature if the effect of direct warming of the skin of the extremities has to be tested, but synchronous readings must be taken of the temperature of the room, of the skin of the extremity, and of the hot-air bath. When these are plotted it is possible to assess the relative importance of the direct effect of external temperature, and of nervous vasomotor reactions, in determining the activity of the peripheral circulation.

Sympathetic activity may thus be inhibited, but it is sometimes possible to go a step further in the investigation and to produce a temporary paralysis of portions of the sympathetic system by means of local anæsthetic drugs. The vasoconstrictor fibres to the legs are readily paralysed by a spinal anæsthetic given so that the upper limit of the anæsthesia reaches the level of the umbilicus, and thermocouples attached at different levels on the extremity will indicate the condition of the peripheral as compared with the more proximal vessels, as well as the total increase in the blood-flow produced by loss of vasoconstrictor tone.

J. C. White² has elaborated a technique for paralysing the sympathetic supply to the brachial plexus by injecting local anæsthetic close to the posterior extremities of the first and second ribs so as to infiltrate the tissue between the pleura and the heads of the ribs through which the sympathetic trunk is passing, and he has used this method as a guide in selecting the cases of vascular disease in the upper extremity which are suitable for sympathectomy. Horner's syndrome is observed when the injection has been correctly performed.

OPERATIONS FOR GANGLIONECTOMY.

1. **Cervico-thoracic Ganglionectomy.**—The object of this operation is the removal of the inferior cervical and the first and second thoracic ganglia, and it is a matter of opinion whether this can be more readily achieved by an anterior or by a posterior approach. There is no doubt that the trunk lies nearer the back of the body, but the posterior operation advocated by Adson³ traverses very vascular muscles and involves the removal of portions of one or, more commonly, two ribs on each side. The sympathetic trunk and its ganglia lie anterior to the thoracic nerves, and the upper part of the inferior cervical ganglion has to be drawn downwards from under cover of the first thoracic nerve, and the complete removal of this ganglion may present considerable difficulty to anyone not particularly familiar with the operation.

The anterior approach, as described by Professor Gask in the Bradshaw Lecture given at the Royal College of Surgeons in December, 1932, in spite of the greater depth of the thoracic trunk from the surface, is a procedure which involves less damage to the tissues, gives a perfect exposure, and, if the anatomy of the part be studied carefully beforehand, will be found to present no difficulty.

2. **Lumbar Ganglionectomy.**—When both lumbar trunks are to be excised the operation is commonly performed by the anterior transperitoneal approach described by Adson.³ Excision of the lumbar trunk on one side only, and

excision of both sides in certain cases in which it may be inadvisable to open the peritoneal cavity, is most conveniently carried out through an incision in the loin, following Royle's technique.⁴

EFFECTS OF SYMPATHECTOMY.

1. Blood-vessels.—

a. Arteries.—It has been mentioned already that removal of the sympathetic produces arterial dilatation, and that the degree of dilatation may be determined by observing the rise in the temperature of the affected part. The accompanying chart (*Fig. 1*) records the temperature of the fingers of both hands after cervico-thoracic ganglionectomy on the right side. The temperature of the right side is constantly higher, and even after immersing the hands in water at 15° C. for thirty-five minutes the right hand could

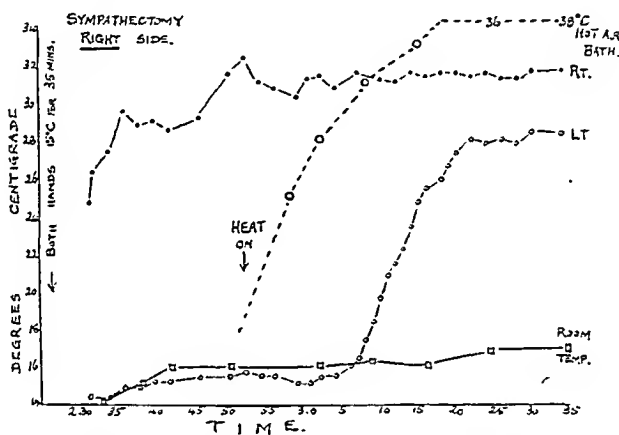


FIG. 1.—Response to heating the body in a patient with normal blood-vessels. Examination performed nine days after cervico-thoracic sympathectomy on right side. The left side shows the normal response. Time in minutes.

not be properly cooled, its temperature rising rapidly after removal of the hands from the cold bath, while the temperature of the left hand remained low and rose normally as the body was heated. The patient from whom these readings were taken was not suffering from any disease of his blood-vessels—the sympathectomy had been done to relieve pain—and he may be regarded as exemplifying the effect of sympathectomy upon normal arteries.

It was shown by Goltz and Freusberg as long ago as 1874 that in the course of time a moderate degree of tonus returns in vessels deprived of their vasoconstrictor nerves; and we have evidence that the results of their animal experiments hold good for man also. One of our patients had cervico-thoracic ganglionectomy performed for a form of arthritis affecting the right arm, the blood-vessels themselves being free from organic disease. Shortly after the operation the temperature of the right hand was considerably raised and the temperature of the hand rose immediately after a period of immersion in cold water. But when she was examined again six months later it

was noticed that the right hand was paler and cooler than the left, and it seemed to her that the temperature of this hand varied more with the external temperature than with the body temperature. After cooling both hands to 15°C . for twenty minutes the body was warmed up in the hot-air bath, and the chart shows that whereas the left hand responded normally to warming the body, the temperature of the right hand remained low, approximating to that of the rather cold room (*Fig. 2*).

It will be seen, therefore, that if after sympathectomy a considerable degree of tonus appears in the denervated vessels, the condition of the patient may be more precarious than it was before the operation, since the vessels are no longer under the control of the nervous mechanism. Lewis's investigations of patients after sympathectomy have led him to the conclusion that there are vasodilator fibres in the sympathetic, and the failure of a limb to warm up some months after operation may be due partly to blocking of vasodilator impulses.

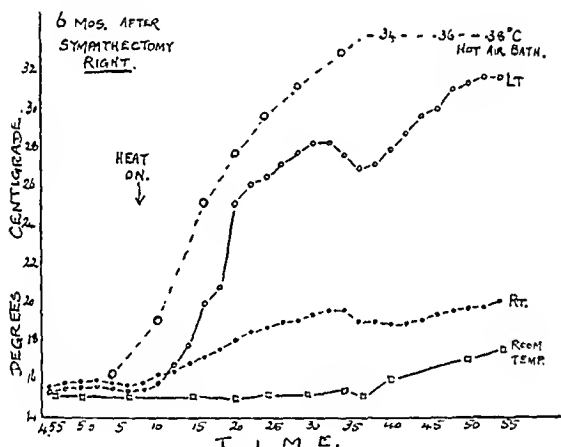


FIG. 2.—Response to heating the body in a patient with normal blood-vessels six months after cervico-thoracic sympathectomy on right side. Normal response on left side. Note more gradual rise of temperature in denervated fingers. Time in minutes.

Cold can produce vasoconstriction reflexly through the vasoconstrictor nerves, but it can also do so by acting directly upon the vessels themselves. The tonus acquired by denervated vessels may therefore be hastened in its onset and increased in intensity by cold. This is in accord with our clinical observations and is of great importance in connection with Raynaud's disease. It has been shown by Lewis that in this condition the fault lies in the small arteries, which are unduly susceptible to cold, and not in their nerve-supply. This conclusion is supported by the result of sympathectomy, for in all our patients who suffered from Raynaud's disease, though their hands were warm after operation, and their limbs showed all the signs of complete sympathetic denervation, coldness and cyanosis of the fingers could be produced by immersing the hands for twenty minutes in water at 15°C . This observation is well illustrated by comparing the chart of the finger temperatures of a patient with normal vessels and that of a patient with Raynaud's

disease, the readings before and after sympathectomy being superimposed (Figs. 3, 4). Prior to all the observations the fingers were cooled to 15°C . for not less than twenty minutes. Before sympathectomy the skin temperatures of both subjects rose as the body was heated, though the sufferer from Raynaud's disease showed a much delayed response. After sympathectomy it was impossible to cool the hand in which the vessels were normal; but in the case of Raynaud's disease the hand failed to warm up in spite of heating the patient's body, the skin temperature rising only very slowly with that of the surrounding atmosphere.

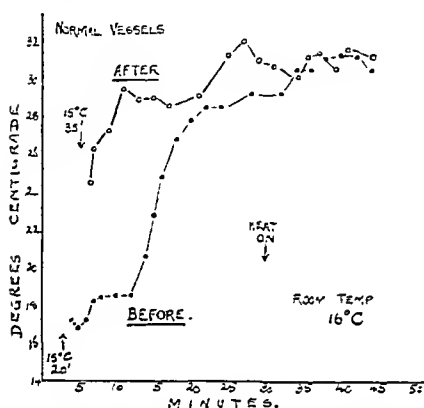


FIG. 3.—Composite chart from same finger of a patient with normal vessels before and after cervico-thoracic sympathectomy. After operation the effect of prolonged cooling of the hand passes off very rapidly.

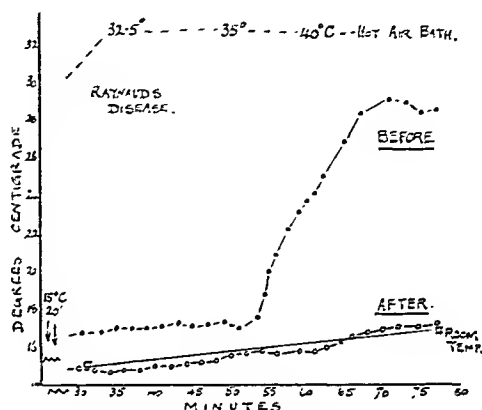


FIG. 4.—Composite chart from same finger of patient with Raynaud's disease before and after cervico-thoracic sympathectomy. Susceptibility of vessels to cold persists after complete sympathetic denervation.

When sympathectomy affords only moderate relief in Raynaud's disease it is sometimes assumed that the operation must have been incomplete. It must be clearly stated, however, that unsatisfactory results may follow complete sympathectomy; and further that the arteries not only retain their susceptibility to cold after sympathectomy, but that in time they may become less responsive to warming the body. It is fortunate that cases vary considerably in severity, and it is only the minority which do not derive considerable benefit from the operation, since under the usual conditions of exposure to a temperate atmosphere the extremities have a higher mean temperature than they had before the operation.

b. Arterioles and Capillaries.—Whereas the arteries are controlled by vasomotor nerves, the minute vessels (arterioles and capillaries), though supplied by nerve filaments, are controlled by chemical bodies, the most important of which is commonly referred to as 'H' substance, whose action upon the minute vessels is the same as that of acetylcholine and histamine. Any tissue injury gives rise to the local production of this 'H' substance, and Lewis⁵ in 1927 brought forward experimental evidence to show that vasodilatation resulting from nervous impulses is due to liberation of 'H' substance at the nerve endings.

Though the minute vessels may be more accurately observed by capillaroscopy, a simple and reliable indicator of the state of these vessels is the colour of the skin. For the first forty-eight hours after cervico-thoracic ganglionectomy the face is flushed, the conjunctiva congested, the hand is hot and red, and the patients sometimes notice a bursting feeling in the extremity. Capillary pulsation in the fingers is frequently observed. But after this initial period has elapsed there is a gradual increase in the tone of the minute vessels, pulsation in them disappears, the conjunctival congestion is lost, and the skin of the denervated arm and hand becomes paler than that of its normal fellow. The limb thus becomes hotter but paler as a result of the operation, and this state persists indefinitely.

The increase in the blood-flow through the arteries ensures the rapid removal of all products of tissue activity, including 'H' substance; and since it is 'H' substance which dilates the minute vessels, the washing away of this body results in constriction of the minute vessels, and pallor of the skin. It is interesting to note here that certain observations we have made of cases of causalgia confirm these views.

Vasodilatation is the most important element in the clinical picture of causalgia—the peculiar burning pain which follows certain peripheral nerve injuries—and on theoretical grounds Lewis suggested that all the manifestations of nerve irritation, including the formation of herpetic vesicles and trophic changes in the skin, would find their explanation in the local action of 'H' substance liberated by antidromic impulses. The following case provides experimental evidence in support of the theory.

V. F., male, 23 years of age, a paper maker, lost the end of his right thumb in a machine accident in December, 1929. The stump healed in three weeks but remained tender, and amputation through the proximal phalanx was performed in March, 1930. Severe causalgia developed involving the distribution of the outer branch of the median nerve, and he was unable to work. In September, 1931, peri-arterial neurectomy of the right brachial artery gave partial and temporary relief, but six weeks later the condition was worse than ever, and there were recurrent crops of herpetic vesicles on the pad of the index finger (*Fig. 5*). In November, 1931, after the man had been unable to work for two years, cervico-thoracic ganglionectomy was performed on the right side. Within forty-eight hours the herpes had completely dried up, and as the red skin of the thenar eminence and index finger became pale, hyperæsthesia disappeared entirely. A tender spot remained in the amputation scar, which proved to be an end-bulb, and since it has been excised the hand has remained free from all pain and hyperæsthesia, and the man is back at work.

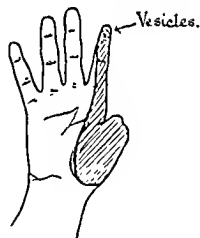


FIG. 5. — Distribution of vasodilatation and herpetic vesicles in causalgia of outer branch of median nerve. Vesicles disappeared, and vasodilatation of minute vessels was abolished after sympathectomy.

We can tell from his specimens and his writings how carefully John Hunter studied vasomotor phenomena, and he would have been interested to see the modern work which is gradually elucidating the mechanisms which control them. Hunter can have known nothing of the functions of the sympathetic system, for though the lateral trunks were accurately described and named by Winslow when Hunter was a child, the work of Claude Bernard,

which marks the beginning of our knowledge of the physiology of the sympathetic, appeared sixty years after Hunter's death.

In his *Lectures on Inflammation*, we read: "I propose to state a few of the facts which throw some light upon the vascular system by showing that there is in vessels a power of muscular action, and that the co-operation of elasticity is also necessary to their function"; and "The colour of an inflamed part is visibly changed from the natural hue, whatever it was, to red. This red is of various hues, according to the nature of the inflammation. The increase of red would appear to arise from two causes, first dilatation of the vessels and secondly the formation of new vessels." Surely John Hunter's enthusiasm for searching out the secrets of human physiology and pathology would be stirred if he were shown that redness of the skin can be produced by irritation of a sensory nerve, and that this skin can be blanched by sympathetic denervation.

2. Secretion of Sweat.—Though pilocarpine will induce sweating in an area completely deprived of its sympathetic nerve-supply, sweating which normally results from exercise or from heating the body is produced reflexly, and is always absent in an area whose sympathetic supply has been severed from the centres in the spinal cord. The line which separates the dry from the sweat-covered skin is an amazingly sharp one, and is quite a reliable method of determining the extent of the sympathetic denervation (*Fig. 6*). The test does not differentiate between areas denervated by section of postganglionic as distinct from preganglionic fibres, since the whole area of skin whose preganglionic supply has been cut will be dry.

Some of our patients have stated emphatically that the secretion of sweat has been considerably more profuse in the areas not affected by the operation. At first we were inclined to regard this merely as an error of observation, the usual amount of sweat being considered excessive when contrasted with the completely dry denervated area. However, the remark has been so frequently made, and it has been possible to observe the profuse secretion so often, that the possibility of compensatory hypersecretion cannot be excluded.

3. Pilomotor Mechanism.—Goose skin may be made to appear as a response to central or peripheral stimulation. If a block of ice is placed between the shoulder-blades, goose skin appears over a wide area as a result of a spinal reflex, and this response is lost after section of any of the sympathetic fibres to the part, whether preganglionic or postganglionic. After

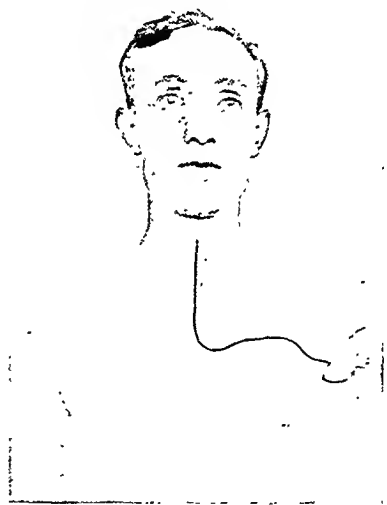


FIG. 6.—Left side of face and neck, left arm and shoulder, and the chest above and to the left of the line completely dry, rest of body bathed in sweat after exercise. Cervico-thoracic sympathectomy, left, by anterior approach.

sympathectomy the area over which this pilomotor response is absent corresponds to the area of skin which remains dry when the body is heated.

But goose skin may also be produced as a result of fairly powerful faradic stimulation of the skin. The response is obtained more readily in some subjects than in others, and we have not used the test extensively because many patients find it distinctly unpleasant or even acutely painful. The importance of the test is that electrical stimulation will produce goose skin even though the preganglionic fibres to the part have been severed; but it is not effective after section of postganglionic fibres when the period required for degeneration of the fibres has elapsed. The test is therefore of value in determining the exact distribution of postganglionic fibres from particular sympathetic ganglia.

4. **Muscular Power.**—Though we have no observations of muscle tone to record, we have noticed that a constant sequela of sympathectomy is weakness of the voluntary muscles in the affected limb. This loss of power may be shown by dynamometer readings, but it is quite obvious without this test, for the patients have been unable for some days after the cervico-thoracic operation to hold a cup while drinking, or to grasp an orange in order to peel it; and a feeling of weight in the arms, and of dragging at the shoulders owing to weakening of the shoulder-girdle muscles, is a common complaint which is relieved by massage and disappears completely in a few weeks. The explanation of this phenomenon is not clear, but it has been assumed that it is due to circulatory changes in the muscles, and certainly not to anything which causes permanent paresis.

5. **Oculomotor Phenomena.**—In a brief but brilliant paper published in 1869, the Swiss ophthalmologist, Friedrich Horner, described a case showing ptosis, myosis, enophthalmos, and diminution of intra-ocular tension which he attributed to paralysis of the cervical sympathetic. He also mentioned the dry warm skin of the face on the affected side, his accurate studies of skin temperature being the first to be made in a clinical case (Fulton).

The preganglionic fibres to the eye may be interrupted by lesions of the brain-stem or cervical cord and by injury to the lower part of the brachial plexus. They are always divided when the stellate ganglion is removed, but in some of our earlier operations in which we merely divided the sympathetic trunk below the stellate ganglion the eye fibres escaped injury, thus showing that they pass out from the cord in the white ramus from the first thoracic segment.

For the first few days after cervico-thoracic ganglionectomy ptosis is very marked, the conjunctival vessels are engorged, and the tension in the eyeball falls to such an extent that accommodation may be impaired. It has occasionally been necessary for patients who have previously worn glasses to have their lenses altered subsequently.

Later the ptosis becomes less marked and the intra-ocular tension rises somewhat, but in none of our patients has restoration of function been complete, even after the lapse of two years, though no appreciable disability results. It is stated that in lower animals complete recovery of the eye eventually occurs, and this difference between man and the lower animals may be of importance, since it would appear that in man the intact efferent

cells and postganglionic fibres cannot function perfectly when the preganglionic portion of the system has been destroyed. It seems probable that this rule may apply to other parts of the sympathetic system in man.

6. Visceral Motor Mechanism.—

a. Colon.—Twenty years ago intestinal stasis was explained by the leaders of surgical opinion on mechanical grounds. The intestine was regarded as a tube subject to obstruction by kinking, with subsequent atony of the portion proximal to the kink. Keith published a paper in 1915 combating this mechanical view and suggesting that symptoms resulted not from atony, but from hypertonicity of those parts which are normally in a state of tonic contraction, thus postulating a disturbance of the neuromuscular mechanism to replace the mechanical theory of intestinal stasis. Keith's conception followed naturally upon the work of Bayliss and Starling on peristalsis, and upon Elliott's investigations into the nerve-supply of the sphincters which Keith himself had described.

We may deduce from experimental data that in the normal individual the antagonistic activities of the sympathetic system and of the cranial and sacral autonomies are in a state of equilibrium. But if the balance is upset so as to produce a preponderance of sympathetic activity in any portion of the alimentary tract, the result will be inhibition of the viscus and increased tonus of its sphincter, with consequent stasis of its content. In spite of Keith's plea to regard the colon as a neuromuscular organ rather than as a sewage pipe, nearly fifteen years had to elapse before surgeons began to apply his ideas to practice. Even then the notion of operating upon the sympathetic system to influence bowel movement seems to have originated in an almost accidental, or at least incidental, observation by Royle, who noted that when he operated on the lumbar trunks for spastic conditions of the legs, certain of his patients who had previously suffered from chronic constipation were relieved of this condition.

In attempting to assess the activity of the sympathetic supply to the bowel, the history of the case and the general condition of the patient may be very helpful. But the most accurate information we are able to obtain at present comes from the careful study of a barium enema, correlated with the patient's sensations, and the effect of spinal anaesthesia upon the barium-filled colon (Scott and Morton⁶).

The remarkable feature as regards the patient's sensations is that pint after pint of barium emulsion can be administered without any complaint of fullness or distension. Seven pints were run into the bowel before the X-ray



FIG. 7.—Megacolon. Barium enema—seven pints given without discomfort.

picture shown in *Fig. 7* was taken, yet the patient, who was only 14 years of age, suffered no discomfort. A spinal anæsthetic was then given so as to produce anæsthesia to the level of the umbilicus. Shortly afterwards the patient complained of what seemed to be a colicky pain, and a wave of peristalsis was seen to pass along the colon, a large quantity of the barium being expelled from the rectum (*Fig. 8*).

It might be expected that the spinal anæsthetic would paralyse all the nerves passing to the bowel, yet the increase in its activity must be taken to show that the predominant effect of the anæsthetic is to cut off the sympathetic inhibition, leaving the intrinsic mechanism of the bowel to act unimpeded.

The sympathetic nerve-supply to the left side of the colon may be inter-

rupted by the operation described by Learmonth and Rankin,⁷ and the effect of this sympathectomy is indicated by the frequency of bowel actions, by the patient's subjective sensations, and by changes in the X-ray findings. The bowels, instead of being opened once a week, will act daily; and whereas large doses of purgative drugs were previously ineffective, a mild aperient will prove sufficient to correct any tendency to constipation.

The patient who had previously lost, or had never experienced, the sensation of fullness in the lower bowel which promotes the act of defæcation, will subsequently acquire such tonus in the bowel that distension with fæces will give rise to the normal sensation; an enema will also produce this feeling of fullness, leading to evacuation of the bowel contents.

Comparison of skiagrams of barium meals and enemata before and after operation will show that the meal is

passed on more rapidly from one section of the intestine to the next; and, instead of the greatly distended colon with a smooth wall, a more contracted gut with many haustrations will be outlined.

b. Bladder.—The nervous control of the bladder musculature involves the balanced activity of the sacral autonomic and sympathetic nerve-supply. The pelvic nerves (sacral autonomies) contain afferent as well as efferent fibres, the efferents being motor to the detrusor muscle and inhibitory to the internal sphincter, this supply being essential to micturition. The sympathetic impulses travel in the hypogastric (presacral) nerves whose efferents are inhibitory to the detrusor muscle and motor to the internal sphincter.



FIG. 8.—Megacolon. Barium enema after spinal anæsthesia. Picture taken a few minutes after *Fig. 7*. Peristaltic wave passing down descending colon has reached sigmoid colon. Barium was being expelled from rectum.

Learmonth⁸ has had the opportunity of stimulating the presacral nerve in patients under spinal anæsthesia, while at the same time the bladder was observed through a cystoscope. He records that stimulation of the presacral nerve causes contraction of the ureteric orifices, increased tonus of the trigone, and contraction of the internal sphincter, the opposite effects being produced by section of the nerve. He also brought forward evidence that stimulation of the sympathetic by adrenalin causes a fall in intravesical pressure.

It must be remarked in this connection that a patient whose presacral nerve has been removed is likely to have transitory increased frequency of micturition, which may be explained by supposing that the desire to micturate, which is due to tension on the muscle of the viscus, can be produced by a smaller volume of urine than was necessary to give rise to this sensation before the nerve was excised.

Although the sympathetic fibres reaching the bladder through the presacral nerve are motor to the internal sphincter, even in the female incontinence of urine does not occur after section of this nerve. Since micturition can be carried out naturally after ablation of the presacral nerve it is clear that only the sacral supply to the bladder is essential, though any disturbance of the balance with preponderance of its sympathetic antagonist may produce symptoms.

The most prominent symptom is an absence of the normal desire to micturate, and in one of our patients who neglected to empty her bladder at regular intervals attacks of retention of urine supervened. After the presacral nerve had been excised the desire to micturate at regular intervals returned, the stimulus being the accumulation of a certain volume of urine in the bladder. If it is allowed that the sympathetic induces relaxation of the detrusor muscle, acting as a brake on this muscle and so preventing a rise of pressure within the bladder as urine collects in it, then the return of the sensation of fullness after interruption of the sympathetic supply will be readily understood.

7. Sensation.—

a. VISCERAL SENSATION.—

i. *Pelvic Viscera*.—Learmonth, in the course of his investigations under spinal anæsthesia, found that though there was analgesia below the tenth thoracic segment, manipulation of the presacral nerve was painful and the pain was referred to the bladder region.

Excision of the presacral nerve has been practised for the relief of pain in carcinoma of the bladder with varying degrees of success, but it is uncertain whether the relief afforded is due to interruption of an afferent path or to relaxation of spasm of the bladder neck. Certain types of uterine pain also have been relieved by the excision of the presacral nerve, but here again the important factor may be interference with efferent rather than with afferent fibres.

ii. *Cardiac Pain*.—The sympathetic nerves mediating cardiac pain are said to be derived from the middle and inferior cervical ganglia; and afferent impulses would pass by them to these ganglia and enter the spinal cord by the white rami communicantes of at least the first three and possibly the first five thoracic nerves, thus accounting for the distribution of the referred pain of

angina pectoris (*Fig. 9*). Leriche claims to have stopped an attack of angina, which occurred while he was actually operating, by injecting novocain into the inferior cervical ganglion.

If this is the path followed by pain which has its origin in the heart, the most rational procedure for its relief is removal of the inferior cervical and first and second dorsal ganglia. In order to avoid the risks of this operation in such patients, White⁹ has advocated blocking the rami of the upper five thoracic nerves on the left side with alcohol, making the injections from the back. I have had the opportunity, with Dr. Geoffrey Bourne, of treating one patient by this method with benefit. According to Cutler's figures operation which includes removal of the inferior cervical and first thoracic ganglia will relieve the pain in about 70 per cent of cases of angina pectoris.

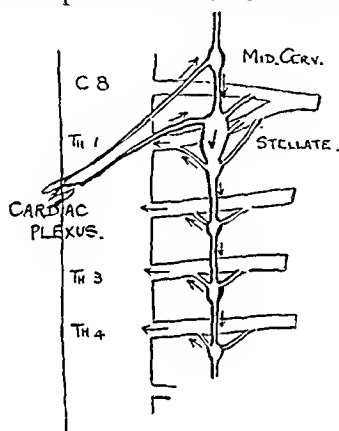


FIG. 9.—Diagram to indicate the path of the pain impulses in angina pectoris.

b. PERIPHERAL SENSATION.—A great deal of confusion in regard to the afferent function of the sympathetic has arisen from the fact that causalgia, though it may persist after various attempts to treat the injured nerve itself, is frequently relieved by sympathectomy. This has led certain clinicians, especially in France, to suppose that causalgia is a neuritis of sympathetic fibres and that the pain in causalgia is conveyed by sympathetic afferents.

It now seems clear, however, that causalgia arises from stimulation of sensory nerve-fibres along which antidromic impulses pass to the area of skin supplied by the nerve, these impulses leading to the local liberation of chemical bodies ('H' substance) which produce vasodilatation and the other trophic changes which characterize the condition. The area of irradiated pain and hyperæsthesia in causalgia is always the site of well-marked vasomotor disturbance, and relief of the symptoms occurs only as a result of treatment directed to the abolition of this vasomotor disturbance. It has already been mentioned that sympathectomy produces such an increase in the blood-flow through the small arteries that the 'H' substance which is responsible for dilatation of the minute vessels is washed away and these vessels become constricted. This constriction of the minute vessels is associated with relief of causalgia, and in this change lies the secret of the success of sympathectomy.

There is evidence to suggest that the characteristic pain may originate in the blood-vessels themselves, but we have been unable to discover anything in our own clinical material to show that the sensation is conveyed along sympathetic pathways. Blood-vessels, especially towards the distal portions of the limbs, are well supplied with nerves, but, as far as we can determine, sympathectomy has no effect upon the pain of arterial puncture. With the help of Dr. Carmichael all forms of sensation were tested in a small group of patients upon whom unilateral cervico-thoracic ganglionectomy had been performed a few months previously, and there was no appreciable difference in the sensation of the two sides. From our own experience, therefore, we must conclude that as sympathectomy is not followed by any impairment of sensation in the limbs, it is unlikely that afferent impulses travel from the limbs to the central nervous system along sympathetic nerves.

REFERENCES.

- ¹ LEWIS, T., *Heart*, 1931, xvi, 33.
- ² WHITE, J. C., *Jour. Amer. Med. Assoc.*, 1930, xciv, 1382.
- ³ ADSON, A. W., and BROWN, G. E., *Surg. Gynecol. and Obst.*, 1929, xlviii, 577.
- ⁴ HUNTER, J. I., and ROYLE, N. D., *Brit. Med. Jour.*, 1925, i, 350.
- ⁵ LEWIS, T., *Heart*, 1927, xiv, 27.
- ⁶ SCOTT, W. J. MERLE, and MORTON, J. J., *Jour. Clin. Invest.*, 1930, ix, 247.
- ⁷ LEARMONTH, J. R., and RANKIN, F. W., *Ann. of Surg.*, 1930, xcii, 710.
- ⁸ LEARMONTH, J. R., *Brain*, 1931, liv, 147.
- ⁹ WHITE, J. C., *Arch. of Neurol. and Psychiat.*, 1929, xxii, 302.

A CASE OF PARATHYROID TUMOUR.

By GEOFFREY KEYNES,

ASSISTANT SURGEON, ST. BARTHOLOMEW'S HOSPITAL;

AND HERMON TAYLOR,

DEMONSTRATOR OF PATHOLOGY, ST. BARTHOLOMEW'S HOSPITAL.

THE condition of hyperparathyroidism, since it was recognized and first treated surgically in 1926, has become almost a common disease. Upwards of fifty examples have been treated by operation and described in the journals of Europe and America, so that it might appear superfluous to describe yet another in this JOURNAL, and the more so because a very full account of the disease was published in its pages by Donald Hunter¹ and A. J. Walton² so recently as October, 1931. In reading the published accounts of the disease, it has appeared to me that attention has perhaps been focused too much upon the changes in the skeleton and too little upon the condition

of the patient as a whole, and it may therefore be worth while to publish a fuller account of a single patient than is usually given, since this patient presents so very complete a picture of the disease, a picture which includes practically every feature that has ever been recorded in hyperparathyroidism, with the addition of one or two that are unusual.

The patient, a male, first became ill at the age of 18 in January, 1925, when he was admitted to the medical wards at St. Bartholomew's Hospital. The initial symptoms were *acute abdominal pain* with *persistent vomiting*, and there was *considerable wasting*. At the same time there were urinary symptoms, with *frequency of micturition*. Renal tuberculosis was suspected, but the urine appeared to be normal, and eventually a small *calculus* was passed by the urethra. A few months



FIG. 10.—Showing widening of right upper alveolar margin.

later (May, 1925) it was realized that the frequency of micturition was due to *polyuria*. At the same time the gastric symptoms continued, and a gastric ulcer was tentatively diagnosed. Proof of this, however, was lacking, and the diagnosis gave way to suspicions of anorexia nervosa or Addison's disease.

The symptoms abated with gastric lavage, and the patient remained fairly well until August, 1926, when there was recurrence of the vomiting, and a second small calculus was passed by the urethra. A slight *albuminuria* discovered at the same time led to a suspicion of chronic nephritis. He was

found to have some degree of *gastroptosis* and *atony of the stomach*, but no further light was obtained as to the diagnosis. In November, 1926, the patient first complained of *pain in the right knee-joint*, with stiffness. The other symptoms were more or less in abeyance, with occasional recrudescence of the vomiting, until May, 1928, when he first came under my own observation with a diffuse bony swelling of the right upper alveolar margin (*Fig. 10*). Many of the peculiarities in the conformation of his skeleton (to be presently described) were then noticed, but their real significance was not appreciated. The swelling in the right maxilla was explored, and a diffuse new growth was diagnosed, the histology being that of a typical *osteoclastoma*, with large numbers of multi-nucleated giant cells.

The patient developed no new features for some time, and was next admitted to hospital with a diffuse swelling of the lower jaw in February, 1930. His general condition appeared then to be unchanged, and exploration of the mandible again produced evidence of new growth resembling osteoclastoma. The maxilla did not appear to have altered, and consequently no further treatment was undertaken.

In July, 1931, the patient began to complain of new symptoms — namely, *aching in the bones* of the limbs, particularly in the right humerus, and troublesome *headaches*. These persisted until the end of that year, when he was more ill in every way. He complained of increased *wasting*, of *dyspnœa*, and of severe *lassitude*. Finally, in January, 1932, he suffered a

spontaneous fracture of the right humerus. By this date a number of parathyroid tumours had been observed and described, so that the diagnosis was no longer in doubt. The patient remained for some time in a cottage hospital, and was not re-admitted to St. Bartholomew's Hospital until June, 1932, by which time the fracture of the right humerus had firmly united, with slight angulation about the middle of the shaft.

At this date the general appearance of the patient was as seen in the photographs (*Figs. 11, 12*) taken two months later, though he was rather more wasted, and there was a more pronounced degree of *muscular atony*.



FIGS. 11, 12.—Showing skeletal deformity and muscular atony.

These pictures show the features summarized below, and it was seen in addition that he had brilliantly *blue sclerotics*.

Head and Face.—Forehead high, broad, and square; the whole head is large in proportion to the body. The right malar region is prominent, corresponding to the enlargement of the maxilla already mentioned; the lower jaw is rounded and thickened.

Thorax.—The chest is narrow and cylindrical, and tapers upwards from the waist. The dorsal spine shows kyphosis and scoliosis. There are numerous bony swellings on the ribs.

Abdomen and Pelvis.—The abdomen is prominent and lax, and the pelvis is wide, with prominent iliac crests.

Lower Limbs.—The great trochanters are prominent and raised. All the signs of bilateral coxa vara are present. There is genu varum, the femurs and tibiae being thickened and curved. There is an enlargement of the upper end of the left fibula. The right knee cannot be fully extended.

Feet.—Tendency to flat feet, but otherwise normal.

Upper Limbs.—Right humerus angulated about its middle. Swellings in the upper parts of the right ulna and the left radius.

Hands.—The appearance of the hands is very peculiar, and the condition does not seem to have been previously described. The chief abnormality is in the terminal joints of the fingers (*Fig. 13*). These are very short and

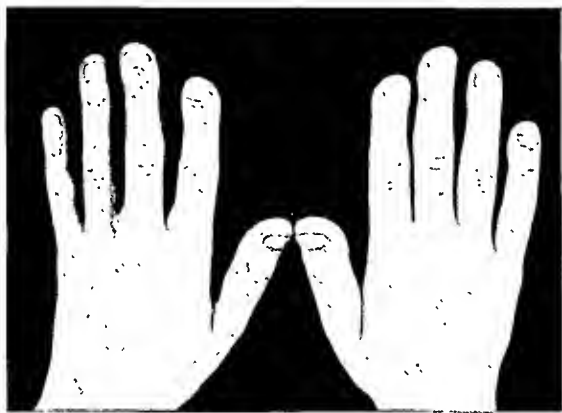


FIG. 13.—Showing abnormality of terminal phalanges and nails.

square, and the nails are correspondingly broad and short. The finger-ends are slightly bulbous, but do not resemble the 'clubbing' of pulmonary osteoarthropathy.

The X-ray examination showed generalized osteitis fibrocystica, with other features corresponding to the deformities mentioned above. Most of these were of the usual type, and do not demand detailed description. The following points, however, are worthy of attention.

Skull (Figs. 14, 15).—This shows very well the thickening of the vault with fluffiness of the bone, and the clear area in the right maxilla corresponding to the tumour previously explored.



FIG. 14.



FIG. 15.

FIGS. 14, 15.—Skiagrams showing thickening of vault of skull, with fluffiness of bone. The clear area in the right maxilla (*Fig. 14*) corresponds to the tumour previously explored.

Humeri (Figs. 16, 17).—This series of radiograms shows the extreme degree of cystic degeneration in the right humerus with the first fracture united, and with a second spontaneous fracture which was sustained after the removal of the parathyroid tumour. The left humerus, however, shows



FIG. 16.

FIG. 16.—Radiogram of humeri, showing cystic degeneration in the right and general fluffiness and osteoporosis in the left.



FIG. 17.

FIG. 17.—Radiogram of right humerus, showing first fracture united and second spontaneous fracture sustained after the removal of the parathyroid tumour.

no cysts, but a general fluffiness and osteoporosis resembling that seen in the cranium.

Hands (Fig. 18).—There is a conspicuous shortening of the terminal phalanges corresponding to the outward abnormality already described.

Right Knee-joint (Fig. 19).—This shows a remarkable degree of metastatic calcification involving the whole of the synovial membrane and the insertion of the quadriceps femoris into the patella. This change was presumably heralded by the onset of pain and stiffness in the joint in November, 1926. The left knee does not show any corresponding change.

The general muscular atony was a conspicuous feature of the patient's condition in 1932. It has been claimed in a recent paper by Max Ballus³

that the presence of atony is a more important diagnostic feature even than a high serum-calcium figure, and he has devised methods for accurately

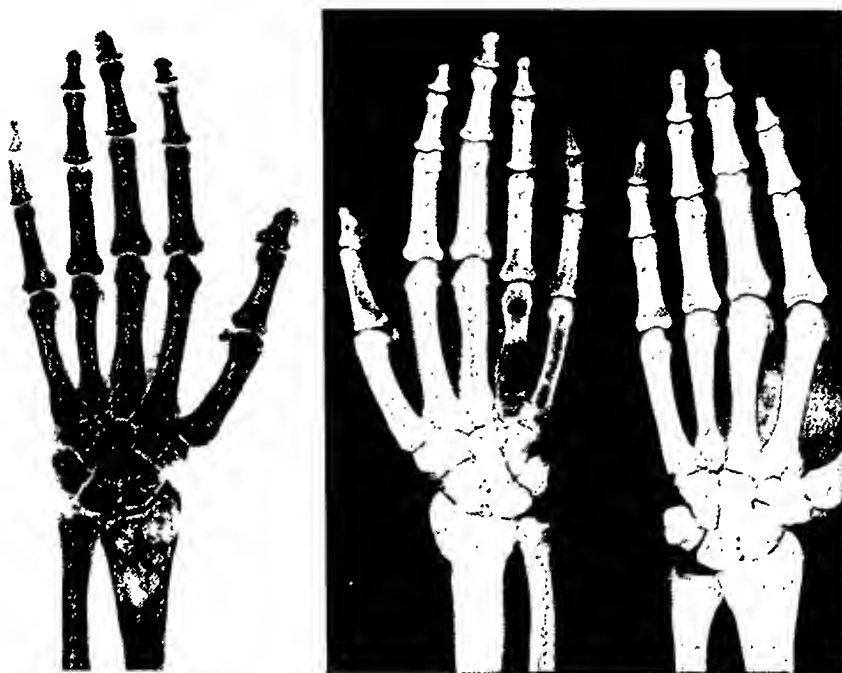


FIG. 18.—Skiagram showing marked shortening of terminal phalanges (with control on right).

measuring its degree. The associated biochemistry of the patient described here was fully investigated by Mr. Hermon Taylor, who gives details in an appendix to this article. The increase in the serum calcium was held to clinch the diagnosis, though no definite tumour could be detected on either side of the neck.

FIG. 19.—Skiagram of right knee-joint showing metastatic calcification involving the whole of the synovial membrane and the insertion of the quadriceps femoris into the patella.

In July, 1932, the neck was explored through the ordinary collar incision. Nothing abnormal was found on the right side. On the left side a cystic tumour was found below and behind the lower pole of the thyroid gland, and was easily removed. This measured 3.5×2.5 cm., and was therefore larger than most of the recorded examples. The greater



FIG. 19.

part of it, however, was cystic, and the amount of secreting tissue it contained was not great. Its histological structure conformed to those already described in this JOURNAL.

The serum-calcium figure fell abruptly after the operation (*see* Appendix), and at the same time the patient's symptoms rapidly abated. His headache disappeared, and he soon expressed himself as feeling better than he had done for years. The premonitory signs of tetany were present for a few days in the form of tingling in the fingers, toes, and nose, but there was no further development. Four months later he was feeling vigorous and active, and the right humerus, originally the most profoundly altered bone in his body, was plainly undergoing re-calcification.

APPENDIX.

By HERMON TAYLOR.

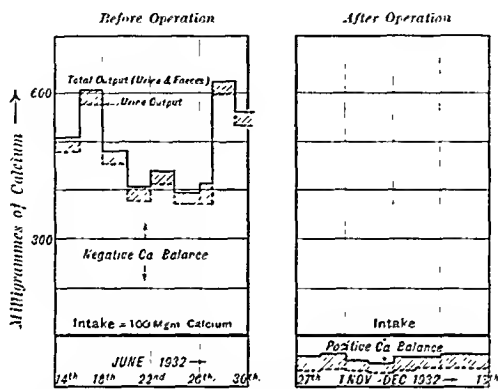
The diagnosis of hyperparathyroidism depends largely on two factors: (1) The X-ray appearances of the bones; and (2) An excessive loss of calcium from the body *by way of the urine*. In the blood a high value of calcium and a low value of phosphorus *usually* accompany this mobilization of calcium from the bones. The level of the calcium in the blood is, however, only a rough indication of the total amount lost from the body, and for this reason, in the early series of cases of osteitis fibrosa, an investigation of the balance between calcium output and intake has been necessary in addition to an estimation of the level of calcium in the serum.

In the cases which have been studied so far, which have appeared clinically and radiologically to be osteitis fibrosa, and in which the serum calcium has been high, an excessive loss of calcium has been found. It appears therefore that, for the routine case of the future, clinical signs of osteitis fibrosa together with a high serum calcium will be sufficient to establish a diagnosis. It must be borne in mind, however, that a normal serum calcium does not negative the diagnosis unless associated with normal calcium output in the urine. It is only in rare cases, which, in spite of a normal serum calcium, still appear clinically to have osteitis fibrosa, that the prolonged and exacting investigation of the calcium balance is necessary, as it is possible for a negative calcium balance to occur with a normal serum calcium.

In the patient here described, calcium and phosphorus balance experiments were performed, and the results are expressed on the four charts (*Figs. 20, 21*). The calcium and phosphorus intake was determined by keeping the patient on a fixed diet of sufficient caloric value with the low calcium content of 100 mgrm. per day, giving him only distilled water to drink. Forty-eight-hourly specimens of urine and four-day specimens of the stools were collected, the periods for the stools being marked off by doses of earmine by the mouth. Estimations of the total calcium and phosphorus in each of these were made and the results plotted on the graphs reproduced (*Figs. 20, 21*).

It will be seen that before operation there was a loss of calcium from the body amounting to roughly 350 mgrm. a day, whereas after removal of the parathyroid tumour there was a gain of calcium of 40 mgrm. a day, indicating that calcification was again taking place in the bones.

The phosphorus balance, on the other hand, showed very little abnormality in the direction of gain or loss before the operation; this is unusual, there being commonly a loss of phosphorus also from the body. The figures for phosphorus balance are, however, much more variable than those for calcium and are of less diagnostic importance than the calcium figures. After operation there was a slight gain of phosphorus.



Dotted line = output in urine. Shaded area = output in faeces.

FIG. 20.—Charts of calcium balance.

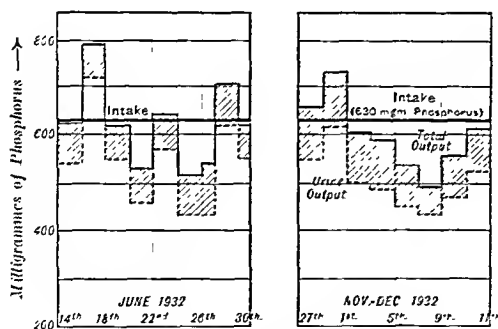


FIG. 21.—Charts of phosphorus balance.

It will be observed from the charts that the increased loss of calcium before operation occurred almost entirely by way of the urine, the amount excreted in the faeces differing very little from that after operation. This is characteristic of hyperparathyroidism, where the calcium mobilized from the bones seems to be excreted almost exclusively in the urine.

In this patient the combination of the X-ray appearances in the bones and the loss of calcium from the body by way of the urine determined the diagnosis of hyperparathyroidism, and it is interesting to note that this relatively large tumour was *not palpable* in the thin neck of the patient. The lower lobe of the thyroid on the opposite side of the neck was abnormally prominent on account of the cervical scoliosis, and this swelling was thought

before operation to be the tumour. The parathyroid adenoma actually lay on the other side of the neck behind the clavicle and sternomastoid.

From this experience it follows that inability to feel the tumour in the neck of a patient showing the clinical appearances of osteitis fibrosa and having a high serum calcium should not prevent one from exploring the neck.

The serum calcium in this case was 14.7 mgrm. per 100 c.c. before operation. Immediately after the operation the value fell to 8.4 mgrm. per 100 c.c., and remained at this level for more than three weeks. It was during this time that the patient experienced the numbness and tinglings of mild tetany. The plasma phosphorus, which was 1.47 mgrm. before operation, rose to 2.6 mgrm. per 100 c.c. at the same time.

Five months after the operation the serum calcium was 11.2 mgrm. per 100 c.c. (high normal), while the plasma phosphorus was 4.4 mgrm. per 100 c.c. (high normal). It is interesting to note that the plasmaphosphatase, which before operation was 0.31 units per c.c., fell to 0.09 units per c.c. (normal) five months after operation.

REFERENCES.

¹ HUNTER, D., and TURNBULL, H. M., *Brit. Jour. Surg.*, 1931, xix, 203.

² WALTON, A. J., *Ibid.*, 285.

³ BALLUS, MAX, *Ann. of Surg.*, 1932, Oct., 478.

POST-TRAUMATIC EPIDERMOID CYSTS OF HANDS AND FINGERS.

By E. S. J. KING,

SURGEON TO OUT-PATIENTS, MELBOURNE HOSPITAL.

Cysts lined, in part, by squamous epithelium occurring on the palmar aspect of the palm (*Fig. 22*) and fingers, and more rarely on the soles of the feet (*Fig. 23*) and on the scalp and face, have been recognized as a clinical entity for over half a century. Although they occur commonly, they exhibit features which are not generally appreciated. They are frequently

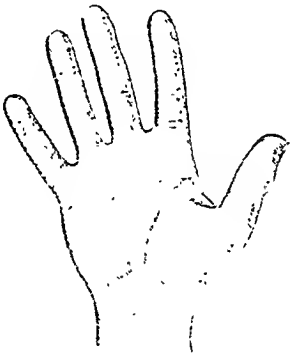


FIG. 22.—Drawing of the hand showing an epidermoid cyst of the hypothenar eminence.



FIG. 23.—Drawing of a foot showing a typical epidermoid cyst.

termed 'implantation cysts' or 'implantation dermoids', a terminology which, in the majority of cases, has but little evidence for its support.

These cysts present problems of special clinical, pathological, and industrial interest and importance. The results of a study of twenty examples of cysts of the hand and fingers together with a brief review of the literature is here given. Their general features may be best exemplified by some typical cases.

CASE HISTORIES.

I. NO HISTORY OF INJURY BUT A BREAK IN THE SKIN.

Case 1.—A male, aged 49, engaged in active work on the permanent way of the railways, sought attention for a swelling in the palm of the left hand. Thirty-five years before this, in the region of the swelling, he had had a 'poisoned' hand, which had burst. After healing, a small 'lump' had remained and continued without any marked variation in size until, three months before seeking attention, it commenced to swell rapidly.

On examination there was seen an irregularly smooth, soft swelling, about $2\frac{1}{2}$ in. in diameter, occupying the region between the base of the ring and little fingers and the proximal crease of the palm (*Fig. 24*).

The cyst was removed under local anaesthesia without difficulty. It was more extensive than the examination suggested and was lobulated, conforming with restrictions imposed on it by the surrounding structures (*Fig. 25*).

It was found to contain a curious fatty substance which differed from sebaceous material, however, in being less greasy, more gritty, and showing a crystalline sheen. On section of the cyst (*Fig. 26*) the contents were found to separate easily from the wall except at one place, where the material was harder, laminated, and closely adherent to the wall. This material was thought to be keratin, and subsequently proved to be so.

Microscopic examination of the wall showed that it was lined in part of its extent by a squamous epithelium without papillae, and throughout the remainder by connective tissue containing spaces originally occupied by fatty material or cholesterol, around which there were giant cells of the foreign-body type. Phagocytic cells and numerous endothelioid cells, fibroblasts, small round cells with occasional polymorphonuclear leucocytes were also present. The cyst contents were mainly typical

cholesterol crystals in one part and keratin in the other. In frozen sections small amounts of fat and fatty acid were demonstrated by their staining reactions to Nile blue, Sudan III, and neutral red. A minute quantity of double-refracting material, which also stained red with Nile blue, was present.

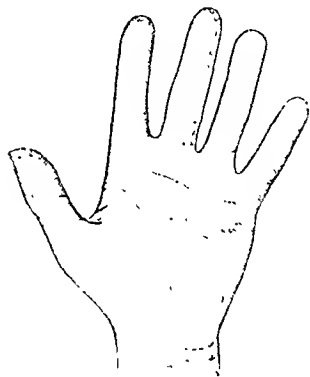


FIG. 24.—Case 1. Drawing of the left hand. The cyst was lobulated and fluctuant. (See *Fig. 25*.)



FIG. 25.—Case 1. Photograph of the specimen removed. The irregularly elongate portion projected, in the palm, towards the wrist and was not obviously palpable before operation. ($\times \frac{1}{2}$.) (See *Fig. 26*.)



FIG. 26.—Case 1. Photograph of the cyst shown in *Fig. 25* after section. It was filled with material resembling sebaceous matter except that it was more gritty and crystalline and less fatty.

Chemical investigation of the cyst contents showed that about half the contents was fatty material and the other half protein. Of the former, 98 per cent was cholesterol, nearly 2 per cent was fat, and the small remainder was fatty acid.

Bacteriological investigation revealed no organisms in the cyst or its contents. Examination of part of the cyst contents by the antiformin method for tubercle bacilli was negative.

II. NO HISTORY OF INJURY.

Case 2.—A male, aged 46, a rat-catcher, was treated for a swelling on the palm of the right hand. He had had this for six years; it was not painful, it had not been tender at any time, and there had been no marked increase in size. He could recollect no special injury to the hand. A rounded swelling about 1 in. in diameter was removed from the region of the base of the ring finger. The cyst was similar to that described in *Case 1*, except that its contour was ovoid.

Case 3.—A male, aged 35, by occupation a pile-driver, sought treatment for a swelling on the palmar aspect of the base of the little finger which he had noticed for eight weeks and which had been becoming larger during that time. There had been no special injury, though he had often bruised his hands. He could remember no injury with any breaking of the skin in this region. A typical cyst $\frac{3}{4}$ in. in diameter was removed.

III. INJURY BUT NO BREAK IN SKIN.

Case 4.—A male, aged 54, who had been attending a medical out-patient department for treatment for paresis of a lower limb which was due to a spinal-cord lesion. He had two swellings, one in the distal part of the first finger, and the other in the proximal part of the ring finger (*Fig. 27*). These caused some inconvenience when using his stick.

The swellings were oval and soft, and appeared to be attached diffusely to the skin. They were not attached to the deeper structures. The patient had noticed the swelling on the first finger since running a thorn into it several months before the time of the examination. As for the other, he was a boot maker by trade and used to run the laces through his fingers always in the same place. The cyst developed at this site. There had never been a wound at this spot. The cyst had been present for nearly a year. Both cysts were found to be typical epidermoid cysts (*Fig. 28*).

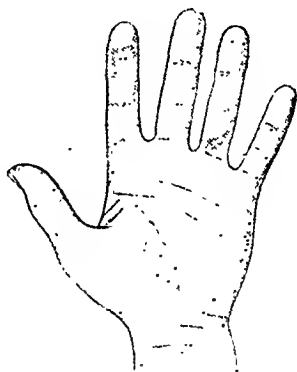


FIG. 27.—*Case 4.* Drawing of the hand showing two cysts. The cyst in the terminal portion of the first finger developed after a perforating injury, and that in the proximal portion of the ring finger after non-perforating trauma. (See *Fig. 28*.)

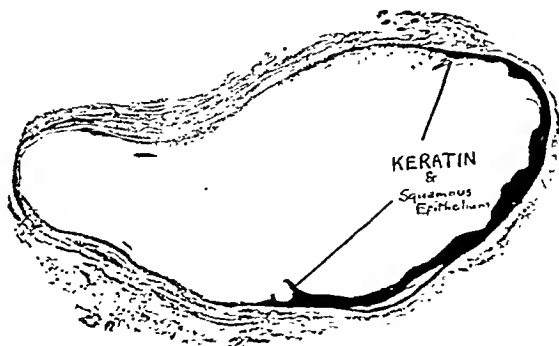


FIG. 28.—*Case 4.* Low-power photomicrograph of the cyst taken from the proximal part of the ring finger (see *Fig. 27*). It is lined in part of its extent by squamous epithelium (without papillae) which is forming keratin (see *Figs. 29, 30*). The remainder is lined by connective tissue containing cholesterol and fat crystals and numerous giant cells. (See *Figs. 29, 32*). ($\times 7$.)

Case 5.—A youth, aged 18, while fielding at cricket was struck over the head of the first metacarpal by a fast-travelling ball. There was immediate pain and swelling. A few days later the swelling measured about $\frac{3}{4}$ in. across and was tender on pressure. This remained, and six weeks later a cyst was removed. It contained old blood, in which there were some cholesterol crystals. The wall was fibrous and there were numerous phagocytic cells and giant cells in the wall. No definite epithelial cells could be found. This cyst was considered to be an early stage of the typical traumatic epidermoid cyst.



FIG. 29.—Photomicrograph of portion of the wall of the cyst (see Fig. 28) showing the junction of the stratified epithelium and the connective tissue lining. ($\times 50$.)

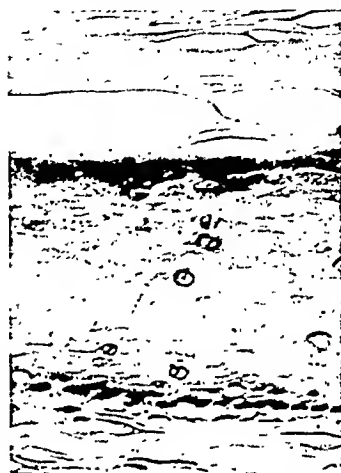


FIG. 30.—High-power photomicrograph showing the character of the squamous epithelium: there are no papillae, a well-developed stratum granulosum, and a large amount of keratin. ($\times 410$.)

Case 6.—A labourer, aged 32, while breaking stones jarred his hand with the handle of his hammer. This remained tender for some time, and within a few days he felt a 'hardness' in the region. He noticed no alteration until ten months later a 'lump' appeared at the site of the injury. This increased in size until eleven weeks later a cyst $1\frac{1}{2}$ in. in diameter was removed. This cyst was similar to the others described.

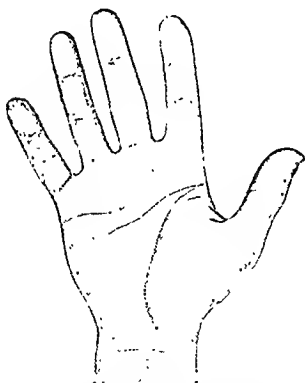


FIG. 31.—*Case 7.* Drawing of the cyst of the hand.

Case 7.—A gardener, aged 42, bruised his hand at the base of the middle finger while shovelling earth. He noticed a small nodule after a few days, but this gave him no trouble and he continued work. Eight months later he noticed that this 'lump' was increasing in size, and nine weeks after observing the increase he sought attention. A rounded cystic swelling about $\frac{3}{4}$ in. to 1 in. in diameter was found (Fig. 31). This was removed, and proved to be a typical epithelium-lined cyst.

IV. INJURY WITH BREAK IN SKIN.

Case 8.—A labourer, aged 38, had his right hand caught between two heavy packing cases. The skin was burst in the palm for $\frac{1}{2}$ in. and there was considerable bruising of the tissues. The wound healed without trouble, but a small lump remained under the scar. Seven months later this nodule began to enlarge, and continued to do so until it was over 1 in. in diameter. It was found to be a cyst similar to those already described.

V. INJURY WITH PERFORATION OF SKIN.

Case 9.—A wharf-labourer, aged 28, received a wound from a sharp hook. The wound healed without complication and he continued work. Five months later a swelling appeared in the subcutaneous tissues at a short distance from the scar. This gradually became larger until it was $1\frac{1}{4}$ in. in diameter. The cyst was similar to those already described.

HISTORICAL.

Epithelial cysts lined by squamous epithelium were first described by Rizet⁴⁶ in 1866. He applied the term 'dermoid cyst', and this view of the nature of the cysts was supported by several observers.^{3, 18, 20, 32, 42, 43} This term may still be found in the literature.²³ Even modern text-books sponsor this view, and state (though not specifically referring to the hand) that such cysts are congenital in origin.

Following Rizet's description, a number of cases were described, and it was noted that the epithelium was not true skin in that it did not possess papillæ, and in 1881 Troquart gave to these cysts the name 'kystes épidermiques'. The contents of the cysts were observed to be like those of sebaceous cysts. Several specimens of cysts of this type were shown before the London Pathological Society, and two of these were called 'sebaceous cysts' by Bowlby⁸ and Poland.⁴³ Villar^{52, 53} and Darier¹³ used the term 'kyste épidermique à contenu sébacée'. This terminology was followed by many writers, and indeed may still be encountered.²² It was found, however, that sebaceous glands are not present in the palm of the hands, and Küster,²⁷ in order to support the sebaceous-cyst hypothesis, postulated that they must occur at times; but extensive investigations have shown them to be invariably absent.

Many of the preceding writers had observed that the development of these cysts was associated with injury, and indeed suggested that epithelium had been carried into the subjacent tissues. The relationship, however, was now emphasized by the use of such terms as 'kystes épidermiques traumatiques',²⁵ 'kyste épidermique consécutif à la traumatisme',^{28, 29} 'traumatic epithelial cyst',²⁴ and 'traumatisehe Epithelevsten'.¹⁷ It will be seen that the recognition of the importance of trauma was becoming general.

In an interesting paper embodying the investigation of three cases, Péraire⁴⁰ in 1892 reviewed the literature and emphasized both the relationship of the cystic development to trauma and also the difference of the cyst content from sebaceous material, and suggested the term 'kyste épidermique traumatique à contenu d'apparence sébacée'.

Much discussion occurred as to the nature of the cysts. Garré,¹⁷ for example, considered that they were due to implantation, which view was accepted by almost all writers, and that various appearances could be found according to whether the displaced epithelium retained its connection with its nerves and blood-vessels. If this last pertained, then papillæ remained: but if the connection were lost, then only squamous epithelium developed from the transplanted cells. Experimental work on the growth of epithelium after transplanatation, particularly that of Kaufmann,²⁶ was quoted in support of the implantation view. On the other hand, Franke¹⁵ pointed out

that a number of cases were not associated with trauma, and deduced that the cysts were due to the late growth of latent embryonal cells.

During this time other hypotheses had been suggested, and the terminology dependent on these used, such as 'pearl tumours' ('cholesteatomata')¹⁹ and 'atheromatous tumours'.²⁸ One writer⁵⁶ spoke of 'heterotopie atheromatous development'. Other terms, which are, however, merely modifications of those already given, are 'epidermal cyst',¹ 'dermal cyst',² and 'kystes dermoïdes traumatiques'.³³ As already stated, they are also frequently referred to as 'implantation cysts'.

During the last thirty years there have been a number of references to these cysts in French, German, Italian, and Russian literature, but there has been a curious silence in English writings, the only statements being those found in text-books or incidental references in papers dealing with general problems.³⁷ Many of the references are to 'traumatic epidermoid cysts', but it is proposed to show that the term 'post-traumatic' would be more satisfactory.

CLINICAL CHARACTERISTICS.

The patients who develop these cysts almost always follow an occupation in which the hands and fingers are liable to trauma, e.g., labourers, carpenters, gardeners, etc. They may develop in individuals who support themselves by means of sticks, e.g., in the case of partial paralysis. Some patients who do not follow arduous occupations develop cysts after injuries, e.g., street accidents.

The type of trauma is of special interest. In the majority of cases following trauma reported in the literature the injury has been a perforating wound or a crush with laceration of the skin. Though such conditions were present in six of the present series, an important observation was that six cases followed non-perforating wounds—jars, non-lacerating crushes, and such traumata.

Absence of trauma in some cases (seven in the present series) presents a serious problem, since cysts identical with the post-traumatic ones occur without history of injury. In many cases, however, the occupation makes the occurrence of traumata almost certain and also the overlooking by the patient of traumata in one particular spot likely. This leaves very few cases in which an injury does not appear to have preceded the cystic development. The cases which form the basis of this paper may be classified as follows:—

No history of injury but a break of the skin ..	1 Case 1
No history of injury	7 including Cases 2 and 3
History of injury without breaking of skin ..	6 including Cases 4, 5, 6, and 7
History of injury with breaking of skin ..	2 including Case 8
History of injury with perforation of skin ..	4 including Cases 4 and 9
Total ..	20 cysts, 19 cases

The age of the patients falls between the second and the seventh decades. The average age in this series was 41; the youngest was 18 years and the oldest 64 years. Johnson²⁴ described a cyst in a patient aged 7½ years. Males are most commonly affected (16 out of 19 cases), but this, like age, is probably subsidiary to trauma.

A most important feature is the latent period between the injury and the onset of the development of the cyst. The latent period may result in the patient's forgetting the injury. In those cases where the injury occurs during work, the necessity for correlating such cysts with the injury for insurance purposes is obvious. The cysts commence to develop any time from a few months up to several years after the injury. This latent period is emphasized by the term 'post-traumatic epidermoid cyst'. Frequently a small nodule or thickening is present at the site of the injury, and this remains until, after some time, the cyst commences to enlarge. Once commenced, the enlargement is rapid, so that a cyst may become $1\frac{1}{2}$ to 2 in. in diameter in less than three months. There is seldom any pain associated with the cysts. Tenderness may be present during the growing period.

Examination of the cyst reveals a rounded, sometimes lobulated, semi-fluctuant or elastic mass occupying some portions of the palmar aspect of the fingers or hand. Cysts of the finger are almost invariably ovoid. They may be adherent to the skin or the subjacent structures, e.g., tendon, or to both. On the other hand, they may be free of these structures, though the size and tenseness may prevent such freedom from being demonstrated.

If they are aspirated, white greasy material will be obtained containing cholesterol crystals in large amount and a little fat.

They rarely become infected, but they may burst externally as the result of further injury, as happened in one case of this series.

Diagnosis is usually easy. If there is a history of injury or a scar, a 'fibroma' round a foreign body must be considered. Usually this is a much harder nodule, but occasionally the diagnosis is not complete until the nodule has been removed. In the absence of any injury, ganglion or tumour of the tendon-sheath must be included in the differential diagnosis. Usually, however, the traumatic cysts are much more superficial than other cysts or nodules.

Removal of these cysts is usually easy. They separate readily from the surrounding tissues.

Complications.—Cysts similar to those occurring in the pulp of the finger may be found also in the phalanx. The relationship of these to the cysts in the soft tissues of the fingers and hand is often not evaluated adequately, as shown by the use of such phrases as 'sebaceous cyst of a phalanx'²²; "at some distant period, a deep puncture wound of the end of the thumb must have carried epithelial cells into the phalanx . . ."; "unfortunately no history of such accident could be obtained". The general interrelationship of the bone and subcutaneous cysts is well discussed by Behrens.⁴

In some cases the cyst of the phalanx follows a perforating injury. In that described by Friedländer¹⁶ the injury was a shell wound, and subsequently Professor Gruber, of Göttingen, demonstrated the presence of epithelial masses occurring in the tissues down to the bone. That the injury resulted in transplantation of tissue here seems to be probable. Even in this case the relationship to the perforating trauma is not absolutely definite, since the cyst did not become apparent until after a second injury (blow by a hammer).

MACROSCOPIC APPEARANCE.

The cysts are usually rounded or ovoid, particularly those in the fingers. Cysts of the palm may be less regular in outline (*see Fig. 25*), having evaginations in various directions whose form is controlled by the surrounding muscles and other tissues.

The wall varies from $\frac{1}{8}$ in. to $\frac{1}{16}$ in. in thickness, usually uniform throughout, but in the larger examples the thickness varies in different parts. Frequently the thickest portion is that nearest the skin, and this part also is tougher owing to keratinous material.

The inner aspect is rough in some parts, smooth in others, depending on whether, after removal of contents, some are still adhering to it (*see Fig. 28*). Laminated keratin is strongly adherent and can only be removed by tearing the wall.

The contents, though superficially resembling sebaceous material, are morphologically, microscopically, and chemically different. Macroscopically they are more gritty and less greasy, contain definite keratin masses, and possess a crystalline sheen.

MICROSCOPIC EXAMINATION.

The cysts consist of a fibrous wall with an inner lining. The fibrous wall varies in thickness and density. In some cases it is merely dense fibrous tissue (*Fig. 32*); often relatively acellular and hyalinized (*Figs. 33, 34*). Thin-walled blood-vessels run for considerable distances in the wall. Nearer the cavity it becomes more cellular and contains numerous cholesterol crystals. Smears of the contents show the typical flat crystals with re-entrant angles.



FIG. 32.—Photomicrograph of portion of a cyst wall showing the structure of the connective-tissue lining. ($\times 70$.)



FIG. 33.—Photomicrograph of portion of the wall of an epidermoid cyst. There is no definite lining and the cyst contents are in direct contact with connective tissue. ($\times 85$.)

EPIDERMOID CYSTS OF HANDS AND FINGERS 37

In the immediate neighbourhood of the cyst wall there are numerous blood-vessels and nerves. Groups of cells which are difficult to identify are

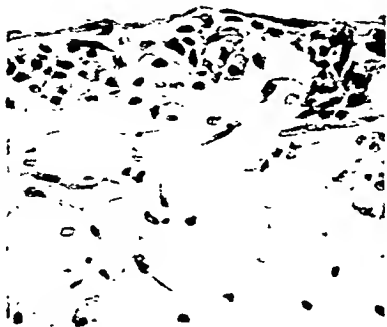


FIG. 34.—Photomicrograph showing the layer of flattened cells on the surface of the connective tissue. ($\times 180$.)

also found (*Fig. 35*). In a few cases I have been able to identify them as epithelial cells. A number of authorities regard them as being groups of sudoriferous gland cells.

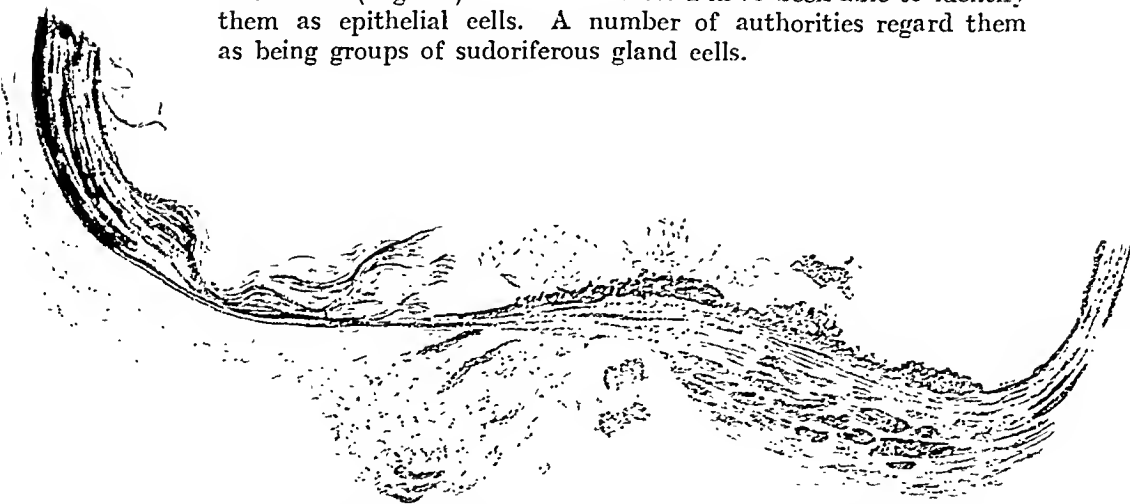


FIG. 35.—Drawing of part of a section of an epidermoid cyst showing portion of the wall lined by squamous epithelium which is forming a large amount of keratin, and the remainder lined by connective tissue containing foreign-body giant cells.

The lining is in part squamous epithelium and in part connective tissue (*see Fig. 28*). The connective-tissue lining shows (in paraffin section) numerous spaces, and surrounding these are foreign-body giant cells of varying sizes and shapes (*Fig. 36*). In some sections polymorphonuclear leucocytes are numerous, though their significance is doubtful. No evidences of bacteria are to be found in sections or contents of the cysts. In some areas the cells of the connective tissue are invading the contents of the cyst (*see Fig. 33*), and such areas show all gradations (*see Fig. 36*) to other parts where the



FIG. 36.—*Case 1.* Photomicrograph of portion of the wall of the cyst showing the cholesterol crystals and giant cells in the wall. ($\times 150$.)



FIG. 37.—Photomicrograph of portion of the wall showing excessive keratin formation. Cysts of this type were, no doubt, the reason for the term 'cholesteatoma' which was applied to these cysts. ($\times 26$.)

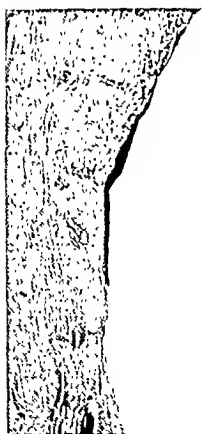


FIG. 38.—Low-power photomicrograph showing an island of squamous epithelium in the wall. ($\times 18$.)

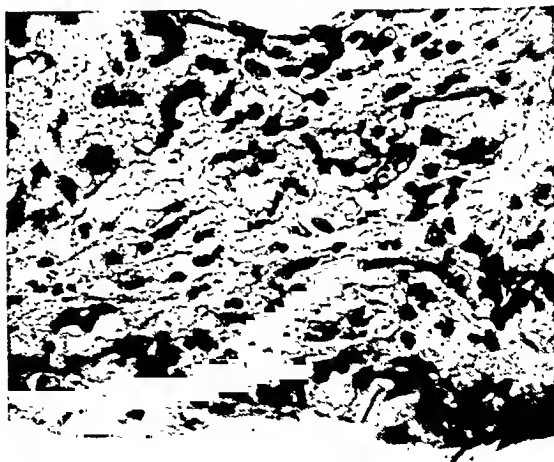


FIG. 39.—Photomicrograph showing the epithelium in a part where it appears to be developing. Some of the cells are columnar in form. ($\times 405$.)

connective tissue is covered by a definite layer of flat cells (*see Fig. 34*). On two occasions I have examined cysts which appeared to be of the same type, as far as could be determined by clinical and macroscopic examination, which possessed no epithelial lining at all, but were lined entirely by the connective tissue.

The well-developed epithelium has a definite basal layer of cuboidal cells, several layers of prickle cells, one or two layers of pigmented cells, and superimposed keratin (*see Fig. 30*). The amount of keratin is often large, giving rise to the use by some of the older writers of the terms 'pearl tumour' or 'cholesteatoma' (*Fig. 37*). Papillæ are very seldom present. They are pictured by Garre¹⁷ and Nicholson,³⁷ and I have observed them in a cyst in the foot. The squamous epithelium may end abruptly (*see Fig. 29*), but it often gradually fades into non-epithelialized areas (*Fig. 38*). Islets of epithelium may be found in various parts of the wall (*Fig. 38*).

All of the epithelium is not typically squamous. Certain of the epithelial islets consist of a flattened squamous epithelium. Some of the cells in such areas are columnar (*Fig. 39*), and some of the epithelium, though only two or three cells thick, produces keratin (*Fig. 40*).

It is usually stated that these cysts are never completely epithelialized, but always show an area of connective tissue where absorption of the epithelial secretion is apparently occurring. This has been my experience in all but two cases, in which every part of the wall examined possessed the squamous epithelial lining.

Nature of the Contents.—The contents of the cysts differ from those of sebaceous cysts and from those completely lined by squamous epithelium. It is usually stated that in sebaceous cysts there is a considerable amount of fat, fatty acids, and a small proportion of cholesterol. In the traumatic cysts the proportion of cholesterol is very high and the amount of fat very small (*see Case 1*). There is also a considerable proportion of keratin.

HYPOTHESIS OF MODE OF FORMATION OF THE CYSTS.

The principal hypothesis accepted at the moment is that postulated by most of the older writers, i.e., implantation of epithelium. This opinion has suffered minor alterations, but the underlying idea remains. Figures



FIG. 40.—Photomicrograph showing the wall with spaces and giant cells in the connective tissue, which is covered by a very low poorly developed squamous epithelium forming keratin. ($\times 150$.)

illustrating the supposed sequence of events are to be found in various papers, e.g., those of Garré¹⁷ and Le Fort.³³ It is possible that this is the explanation for some cases but not for all. The claim for this view depends on three observations: (1) A perforating wound sometimes precedes the development of the cyst; (2) The cyst is lined by squamous epithelium; (3) Experimental implantation of skin results in the formation of an epithelium-lined cyst. The arguments which may be raised against this view are:—

1. In many cases the injury is not of the perforating type, and a considerable number of cases do not give a history of trauma. It is unlikely that perforating injuries would be overlooked, but smaller, non-perforating, possibly jarring injuries without loss of continuity of the surface might be forgotten.

2. The presence of the latent period between the injury and the development of the cyst makes it improbable that actual implantation of the surface epithelium occurred. Implantation of tissue usually results in the immediate formation of a mass (often cystic) arising by growth of the implanted tissue. Animal experiments²⁶ result in the formation of a cyst in a comparatively short time. While considerable periods, even years, may intervene in the type of case here described between the injury and the development of the cyst.

3. The method of development is also peculiar. After the latent period the cyst becomes large relatively rapidly. In cases of known implantation the cysts come to a definite size and then grow more slowly or may even regress.

4. The epidermoid nature of the wall does not necessarily support the implantation hypothesis.

Two types of epithelium may be found: (1) A squamous epithelium without papillæ—hence the term ‘epidermoid’ as applied to these cysts; and (2) Squamous epithelium with papillæ, which is uncommon.

A simple squamous epithelium arises so readily from epithelia of various types that the mere presence of tissue showing squamous characters does not indicate the origin from skin. Chiari has shown that certain subcutaneous cysts arise from the glands in the skin. Even the squamous epithelium with papillæ is not inevitably derived directly from the overlying skin. However, even if those cysts which are so lined are regarded, from the nature of the epithelium, as due to implantation, such cases do not always give a history of perforating injury.

Thus the evidence, at first sight so strongly in favour of the implantation hypothesis, actually is feeble and does not uphold the view.

Many curious forms of epithelium are found in these cysts. Usually it is stated that the squamous epithelium becomes flattened by pressure—a process, in view of the growth of cells under considerable pressures, and because such pressures are hydrostatic, difficult of explanation. On the other hand, the presence of columnar cells in some parts (*see Fig. 39*) and the small areas of well-developed epithelium tailing off into non-epithelialized parts of the wall without any evidence of degeneration in these areas, suggests that the epithelium has arisen secondarily. This is also supported

by the appearance of the epithelium in places where it is poorly developed (*see Figs. 38, 40*). The problem, then, is the origin and nature of the epithelium.

Origin of Epithelium.—There are three possible modes of epithelial development: (1) By implantation of whole skin; (2) By separation of some few cells of the deeper layers of the epithelium; (3) By development from some other source.

1. This method of formation has been considered already.

2. This suggestion is similar to that of Garré in which the implanted epithelium has lost its connection with nerves and vessels. It will not explain those cases, however, in which the injury is slight or where it is an irritation rather than definite injury. It would surely require considerable force to project epithelial cells for several millimetres, in some cases, through the dense fibrous connective tissue.

One curious feature of epidermoid cysts is the presence of a definite layer of connective tissue between the cyst and surface epithelium, the papillæ not being distorted by presence of the cyst. This suggests that the cyst commences to form in the deeper layers of the dermis or in the subcutaneous tissues.

3. The development of the epithelium from sudoriferous glands is a satisfactory theory explaining most of our observations.

In 1905 Pels-Leusden³⁹ made the suggestion that the epithelium did not arise from the surface epithelium, and concluded that this epithelium probably arose from the cells of the sudoriferous glands and their ducts. (The absence of sebaceous glands from the palm of the hand has already been noted.)

Examination of the walls of cysts shows that the squamous epithelium frequently develops first, or at least is to be found, in that part of the wall near the skin, and examination of the wall in this region shows some sudoriferous glands. Le Fort³⁹ mentions the occurrence, in the neighbourhood of the wall, of cells which, although he regards them as displaced squamous epithelium, are probably of gland origin. Such groups of cells are to be found in the neighbourhood of the walls of most cysts. Glands and cell groups occur mainly in that portion of the wall of the cyst which is near the skin. In two cases of this series sudoriferous glands were particularly numerous.

The mode of development is probably as follows: The injury results in a hæmatoma,⁹ or in some cases probably a chronic inflammatory focus (traumatic in origin) resulting in the formation of a cyst (*Case 5*). This cyst at first contains fluid and degenerating material. It may remain in this form, or even retrogress slightly, during several months. The presence of the cyst, possibly the irritation due to its contents, causes proliferation of the sudoriferous glands in the neighbourhood. Some of these reach the surface of the cyst, where they proliferate—first as columnar cells, then they rapidly stratify and keratinize. An alternative is that some of the cells of the glands are incorporated in the original injured area, and grow, thus forming a cyst. At present it is not possible to prove either suggestion.

It is when the epithelium forms that the cyst commences to grow in size, probably owing to the accumulation of the products of the epithelial activity.

It is probable that with further information it may be necessary to modify the view of the origin of the epithelium, but the important feature is that the cysts are not always due to 'implantation' of skin.

SUMMARY.

1. Cysts of the palmar aspect of the hand and fingers arising after trauma and lined by squamous epithelium are common.

2. The trauma is not necessarily of perforating character; jarring injuries produce a similar result. In a certain number of cases no history of injury is obtainable.

3. There is usually a latent period between the injury and the development of a cyst.

4. The term 'post-traumatic epidermoid cyst' is suggested as emphasizing this feature.

5. The cyst often develops rapidly once it commences.

6. The cysts are benign and easily removed.

7. The squamous epithelium lining them, in the majority of cases, does not arise by implantation from the skin.

REFERENCES.

- ¹ BARKER, A. E., *Brit. Med. Jour.*, 1885, ii, 871.
- ² BARKER, A. E., *Trans. Pathol. Soc. Lond.*, 1886, xxxvii, 478.
- ³ VAN BECELAERE, *Jour. de Sci. méd. de Lille*, 1887, ii, 222.
- ⁴ BEHRENS, A., *Virchow's Arch.*, 1931, cclxxx, 145.
- ⁵ BEUL, A. P., *Moskowski med. Jnr.* (Moskva), 1927, vii, No. 3, 35.
- ⁶ BOPPE, *Bull. et Mém. Soc. Anat. de Paris*, 1923, xciii, 143.
- ⁷ BOUCHET, P., *Ibid.*, 1904, lxxix, 521.
- ⁸ BOWLEY, A. A., *Trans. Pathol. Soc.*, 1882, xxxv, 399.
- ⁹ BRIGGS, F. M., *Boston Med. and Surg. Jour.*, 1895, cxxxiii, 7.
- ¹⁰ CAMUS, M., and ERTZBISCHOFF, P., *Bull. et Mém. Soc. Anat. de Paris*, 1904, lxxix, 706.
- ¹¹ CARROSSINI, G., *Gior. di. med. ferror.* 1921, i, 20.
- ¹² CHAPUT, and GUILLEMOT, A., *Bull. et Mém. Soc. Anat. de Paris*, 1898, lxxiii, 741.
- ¹³ DARIER, J., *Ibid.*, 1889, lxiv, 50.
- ¹⁴ FRANKE, F., *Wien. klin. Woch.*, 1890, iii, 696.
- ¹⁵ FRANKE, F., *Zentralb. f. Chir.*, 1898, xxv, 369.
- ¹⁶ FRIEDLÄNDER, C., *Ibid.*, 1930, lvii, 209, 1854.
- ¹⁷ GARRÉ, C., *Beitr. z. klin. Chir.*, 1894, xi, 1.
- ¹⁸ GIBIER, P., *Soc. Anat.*, 1881, Nov. 18. Quoted by Péraire.
- ¹⁹ GROSS, *Soc. de Méd. de Nancy*, 1884, Jan. 15. Quoted by Le Fort.
- ²⁰ GUERMONPREZ, F., and TOISON, J., *Jour. de Sci. méd. de Lille*, 1885, vii, 405.
- ²¹ HAMMANN, *Deut. Zeits. f. Chir.*, 1930, cccxiii, 308.
- ²² HARRIS, R. I., *Jour. Bone and Joint Surg.*, 1930, xii, 647.
- ²³ JAUREGUI, P., *Bol. y trab. de la Soc. de cir. de Buenos Aires*, 1929, xiii, 233.
- ²⁴ JOHNSON, R., *Trans. Pathol. Soc.*, 1893, xlv, 146.
- ²⁵ JONNESCO, *Bull. et Mém. Soc. Anat. de Paris*, 1888, lxiii, 940.
- ²⁶ KAUFMANN, E., *Virchow's Arch.*, 1884, xeviii, 236.
- ²⁷ KÜSTER, *Ein chirurgisches Triennium*, 1882, 251, Berlin. Quoted by Péraire.
- ²⁸ KUMMER, E., *Arch. prov. de Chir.* 1892, i, 357.
- ²⁹ LABOUGLE, J., *Bull. Soc. d'Anat. et Phys. de Bordeaux*, 1889, x, 141.
- ³⁰ LABOUGLE, J., *Jour. de Méd. de Bordeaux*, 1889-90, xix, 153.
- ³¹ LABOUGLE, J., *Arch. de Méd. de Bordeaux*, 1894, xxiii, 47.

- ³² LALITTE, C. J., *Thèse de Nancy*, 1885. Quoted by Péraire.
- ³³ LE FORT, R., *Rev. de Chir.*, 1894, xiv, 1013.
- ³⁴ MACHAN, B. J., *Vestnick Khirurgii. Moskva*, 1928, xiii, Nos. 37-38, 158.
- ³⁵ MÉTIVET, G., *Presse méd.*, 1919, xxvii, 192.
- ³⁶ MÉTIVET, G., *Semaine d. Hôp. de Paris*, 1926, iii, 373.
- ³⁷ NICHOLSON, G. W., *Guy's Hosp. Rep.*, 1922, lxxii, 402.
- ³⁸ ORBACH, E., *Med. Wch.*, v, 269.
- ³⁹ PELS-LEUSDEN, *Deut. Med. Woch.*, 1905, Aug. 24, No. 34, 1340.
- ⁴⁰ PÉRAIRE, M., *Rev. de Chir.*, 1892, xii, 874.
- ⁴¹ PÉRAIRE, M., *Bull. et Mém. Soc. Anat. de Paris*, 1895, lxx, 607.
- ⁴² POLAILLON, *Bull. Soc. de Méd. de Paris*, 1885 (1884), xix, 125.
- ⁴³ POLAND, J., *Trans. Pathol. Soc. Lond.* 1884, xxxv, 399.
- ⁴⁴ REVERDIN, J. L., *Rev. méd. de la Suisse Rom.*, 1887, vii, Nos. 3 and 4.
- ⁴⁵ REVERDIN, J. L., and MAYOR, A., *Ibid.*, 1889, ix, 99.
- ⁴⁶ RIZET, F., *Arch. gén. de Méd.*, 1866, ii, 615. Quoted by Péraire.
- ⁴⁷ SPASOKUKOTSKI, S. I., *Khirurgia*, 1898, iii, 431.
- ⁴⁸ STABB, E. C., *Trans. Pathol. Soc. Lond.*, 1891, xlii, 314.
- ⁴⁹ THOMAS, W. T., *Liverpool Med.-Chir. Jour.*, 1895, xv, 416.
- ⁵⁰ TIKHOFF, P., *Med. Obozi*, 1895, xliii, 635 ; 1896, xlv, 15.
- ⁵¹ TROQUART, R., *Bull. Soc. d'Anat. et Physiol. de Bordeaux*, 1881, ii, 157 ; *Jour. de Méd. de Bordeaux*, 1881. Quoted by Péraire.
- ⁵² VILLAR, F., *Bull. et Mém. Soc. Anat. de Paris*, 1887, lxii, 591.
- ⁵³ VILLAR, F., *Bull. Soc. clin. de Paris* (1886) 1887, x, 58.
- ⁵⁴ WEGNER, *Deut. Zeits. f. Chir.*, 1898, l, 201.
- ⁵⁵ WÖRZ, A., *Beitr. z. klin. Chir.*, 1897, xviii, 753.
- ⁵⁶ ZMIGRODZKI, K. I., *Protok. i. trudi Russk. chir. Obsk.*, Pirogavia, 1889-90, viii, 205.

AN INVESTIGATION OF 742 CASES OF HÆMATURIA.

BY R. K. DEBENHAM,

SURGEON TO OUT-PATIENTS, QUEEN'S HOSPITAL, BIRMINGHAM;
LATE SURGICAL FIRST ASSISTANT, THE LONDON HOSPITAL.

A STUDY has been made of 742 consecutive cases of hæmaturia which attended the Genito-urinary Department of the London Hospital under the care of Mr. Hugh Lett during the years 1924-30. The majority were treated entirely in the Out-patient Department; the remainder were admitted to the wards either directly or after a preliminary investigation in the Out-patient Department.

The object of this study was to find out the various causes of hæmaturia, and to discover, if possible, what happened to those cases that were unexplained in spite of investigation. Letters were written to all patients in whom the bleeding had not been satisfactorily explained, to find out what had happened to them, and, if necessary, they were asked to attend for further investigation.

The paper is divided into three main sections: (1) A general consideration of the causes of hæmaturia, sex incidence, age incidence, severity of the bleeding, and the common causes thereof. (2) A consideration of the various conditions giving rise to hæmaturia; the relative importance of the main symptoms is evaluated. (3) Consideration of the unexplained cases, with the results of the follow-up investigation.

Patients in whom red blood-corpuscles could be found only on microscopical examination of the urine are not included in the investigation. The presence or absence of hæmaturia was noted when the history was taken. The routine investigation included the clinical testing of the urine in the department, and examination of twenty-four-hourly and sterile specimens in the clinical laboratory whenever necessary. An X-ray examination of both kidneys and bladder was made as a routine. Every case was examined with the cystoscope unless there was some definite contra-indication. Urethroscopy was performed where necessary. If the case required it, the patient was admitted into hospital for pyelography, renal efficiency tests, and any other necessary investigations. It must be mentioned that these cases attended this department before intravenous pyelography was available for investigation.

The two sexes have been studied separately according to the severity of the hæmaturia and grouped as follows:—

Group A.—When hæmaturia was the presenting symptom.

Group B.—When hæmaturia was an important symptom.

Group C.—When hæmaturia had occurred, but was slight and formed merely an incident in the history.

Fig. 41 is a chart to illustrate these groups and to indicate the various causes of hæmaturia.

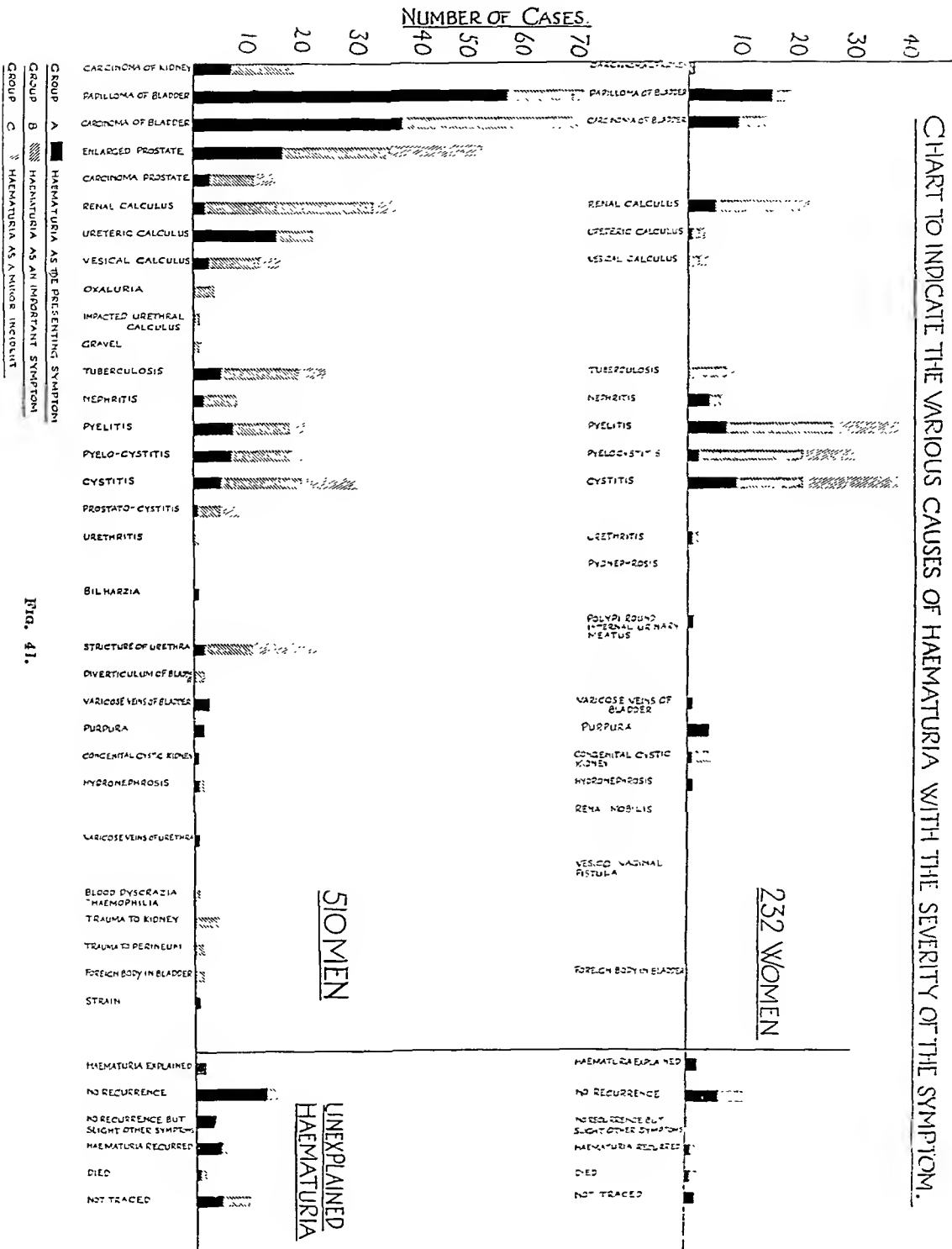


FIG. 41.

Conclusions drawn from the Chart.—Hæmaturia is much more common in men than in women; the proportion is more than two to one.

Inflammatory conditions of the genito-urinary tract are the commonest causes of hæmaturia in women, whereas papilloma or carcinoma of the bladder is the commonest cause in men.

In older men, from 50 upwards, the prostate is a very common cause of hæmaturia—either from simple enlargement with congestion of the veins over it, or much less commonly from carcinoma of the prostate.

The inflammatory conditions occur earlier in life, especially between the ages of 20 and 40, the incidence of this trouble rising rapidly from the age of 20 onwards. In the female sex this is largely accounted for by child-bearing. Pyelitis of pregnancy, cystitis due to infections resulting from parturition, a subsequent cystocele, together with the short female urethra which predisposes to ascending infection, constitute important factors which favour the occurrence of inflammatory troubles in women between these ages.

On the other hand, if a man complains of hæmaturia as the presenting symptom there is almost a 50 per cent chance that it is due to either a papilloma or a carcinoma of the bladder—in this series, 97 cases of hæmaturia as a presenting symptom were due to this cause, and 100 cases were due to other causes.

HÆMATURIA AS THE ONLY SYMPTOM.

Men.—In the case of a man, if hæmaturia is the *only* symptom, there is a two to one chance that it is due to a papilloma or carcinoma of the bladder. There were 65 cases of hæmaturia as an only symptom; of these 44 were due to a papilloma or carcinoma of the bladder, and 21 were due to other causes, and, in spite of full investigation, only 9 of these cases could be explained completely when investigated.

Of the 12 unexplained cases, 1 was proved later to be due to renal calculus, 6 remained perfectly well, 1 died eight years later of carcinoma of the prostate, and 3 said that they had had recurrences of the hæmaturia on one or two occasions after attending hospital. These, however, were perfectly well when written to one year, four and a half years, and five years later, and though the hæmaturia had been 'unexplained' at the time, the patients were none the worse in consequence.

Of the 9 explained cases the causes of hæmaturia were: Prostatic enlargement 3 cases, pyelitis 2, carcinoma of prostate 1, renal calculus 1, varicose veins of bladder 1, varicose veins of urethra 1.

Women.—There were 10 cases of hæmaturia as an *only* symptom in women: Papilloma of bladder 3, renal calculi 2, nephritis 1. And of 4 unexplained cases, 2 later passed stones (though X-ray examination had been negative), 1 remained perfectly well, and 1 could not be traced.

HÆMATURIA IN THE VARIOUS DECADES OF LIFE.

The graph (*Fig. 42*) indicates the relative frequency of the various causes of bleeding at the different age periods. The common causes of hæmaturia are grouped together thus: (1) *Inflammatory causes* (pyelitis, pyelo-cystitis,

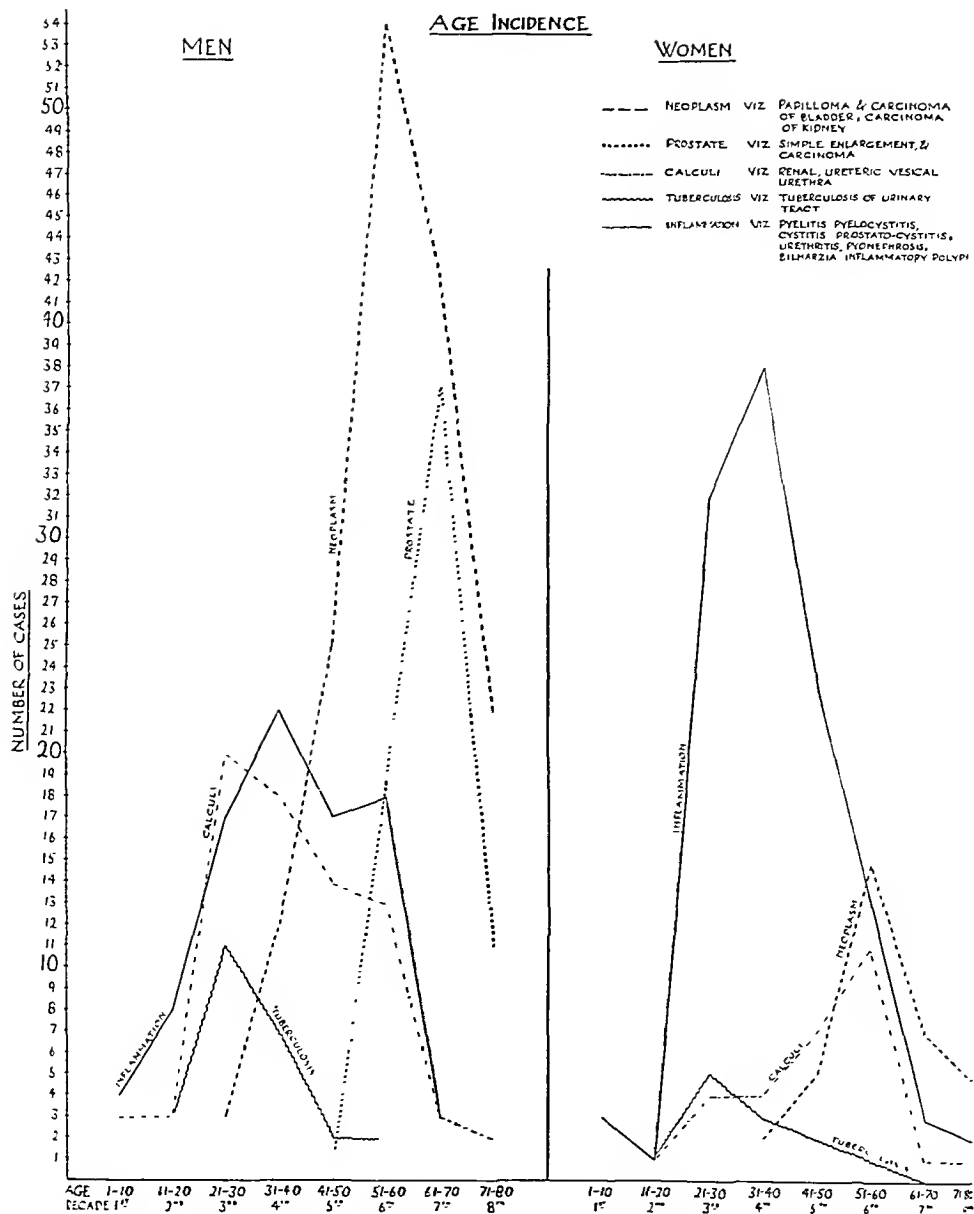


FIG. 42.—Graph indicating the relative frequency of the various causes of bleeding at the different age periods.

cystitis, prostatitis, urethritis, pyonephrosis, etc.); (2) *Calculi* (renal, ureteric, vesical, and urethral); (3) *Tuberculosis*; (4) *Neoplastic* (papilloma and carcinoma of the bladder and carcinoma of the kidney); (5) *Prostatic* (simple enlargement and carcinoma).

The graph shows that :—

1. Hæmaturia during the first two decades of life is rare.
2. In both sexes inflammatory causes and calculi are the commonest causes of hæmaturia during the first four decades of life.
3. Inflammatory causes in females reach their peak in the fourth decade of life; there were 38 cases during this decade.
4. Neoplastic conditions begin to be common during the fifth decade of life, and reach their peak, in both sexes, during the sixth decade. In the sixth decade they form the commonest causes of hæmaturia in both sexes.
5. In the sixth and seventh decades of life the prostate is a very common cause of bleeding.
6. In every decade of life hæmaturia is more common in males than females; this preponderance is most marked after the age of 50, and is then especially due to the greater frequency with which bladder growths occur in men than in women.

An analysis of the complete list of cases shows the following :—

First Decade (age 1 to 10).—Calculi and pyelitis were almost the only causes of bleeding during the first decade of life; pyelitis or cystitis or both being the only causes in girls.

Second Decade (age 11 to 20).—

Boys.—Inflammatory conditions (pyelitis and cystitis) are the commonest causes of hæmaturia in boys during the second decade; tuberculosis and calculi come next.

Girls.—Hæmaturia during the second decade is rare, there were only 5 cases; 2 were caused by stone, 1 by tuberculosis, 1 by pyelitis, and 1 by a foreign body in the bladder.

Third Decade (age 21 to 30).—

Men.—Pyelitis and cystitis, tuberculosis, and stone are the commonest causes (in this order). The only neoplastic conditions that occurred during this decade were 3 cases of papilloma of the bladder; there were no cases of neoplasm during the first two decades of life, and the condition is rare in the third decade.

Women.—Pyelitis and cystitis were much the commonest causes of hæmaturia; then followed tuberculosis and stone.

Fourth Decade (age 31 to 40).—

Men.—Inflammatory conditions (cystitis and pyelitis) are still the commonest causes of hæmaturia, followed by stone, papilloma of the bladder, and tuberculosis respectively. This was the first decade in which there were any cases of malignant disease (2 cases of carcinoma of the bladder).

Women.—Inflammatory conditions (pyelitis and cystitis) are again much the commonest causes of bleeding; neoplasm does occur, but is rare.

Fifth Decade (age 41 to 50).—

Men.—For the first time neoplasm becomes the commonest cause of hæmaturia (papilloma and carcinoma of the bladder). Stone and inflammatory

conditions (pyelitis and cystitis), which are about equally frequent, are less common causes, while tuberculosis is rare.

Women.—Inflammatory conditions (pyelitis and cystitis) are still much the commonest etiological factors, stone and growths of the bladder being much less frequent.

Sixth Decade (age 51 to 60).—

Men.—Neoplasm, especially papilloma of the bladder, is the commonest cause; in this decade it is followed by the prostate, with inflammatory conditions third.

Women.—For the first time neoplasm becomes the commonest cause of hæmaturia (especially papilloma of the bladder); then come inflammatory conditions (cystitis and pyelitis), and, thirdly, stone; all three of these conditions occur with about the same frequency in this decade.

Seventh and Eighth Decades (age 61 to 80).—

Men.—Enlarged prostate and carcinoma of the bladder are by far the commonest causes of hæmaturia during these decades; then follow papilloma of the bladder, carcinoma of the prostate, carcinoma of the kidney, and stricture of the urethra with secondary infection. It will be seen, therefore, that the two most important examinations are rectal examination and cystoscopy.

Women.—Papilloma and carcinoma of the bladder are the commonest causes; the condition is much less common in women than in men.

CAUSES OF HÆMATURIA.

The following table shows the relative frequency of the various causes of hæmaturia.

			TOTAL Per cent	MEN Per cent	WOMEN Per cent
Carcinoma of kidney	2.5	3	Less than $\frac{1}{2}$
Papilloma and carcinoma of bladder	23	27	14
Calculi (renal, ureteric, vesical, urethral)	14.5	15	Nearly 13
Tuberculosis..	5	Nearly 5	Just over 5
Pyelitis	Inflammation	..	25.5	16	46.5
Pyelo-cystitis					
Cystitis					
Urethritis					
Prostato-cystitis					
Pyonephrosis					
Bilharzia					
Prostatic enlargement	7	10	—
Carcinoma of prostate	2	Nearly 3	—
Stricture	3	4	—
<i>Unexplained Hæmaturia</i>	9	7	12
SUB-DIVISIONS	Hæmaturia subsequently explained		0.5		
	No recurrence of hæmaturia		3.5	Nearly 3	5
	No recurrence of hæmaturia but slight other symptoms		0.8		
	Hæmaturia recurred		1.3		
	Died subsequently		0.5		
	No reply received		2.3		

Cystoscopy during an attack of hæmaturia is so valuable that if a patient comes to hospital during the attack of bleeding he should have a cystoscopic examination forthwith, unless there is some definite contra-indication such as

acute cystitis. Only in this way can the source of the hæmorrhage be determined accurately; a carcinoma of the kidney may be detected thus, or some other lesion causing bleeding from one kidney. Or if blood is seen coming from both ureteric orifices, the lesion is bilateral, and probably of medical rather than surgical interest.

Not infrequently hæmorrhage is so great that it is impossible to obtain a view of the bladder; under these circumstances rest in bed is a most valuable method of stopping the hæmaturia. Other aids are the irrigating cystoscope or liquid paraffin as a medium. Sometimes a cystogram is useful.

Marked bleeding from the bladder, or a very 'dirty' bladder, which takes a long time to wash clean, is of course strongly in favour of a bladder lesion rather than of a lesion of the kidneys, and is especially suggestive of carcinoma vesicæ or tuberculosis.

The various causes of hæmaturia are now considered *seriatim* in the order shown on the chart.

Carcinoma of the Kidney (18 cases: 17 men, 1 woman. Average age 56).—A surprisingly small number of patients proved to be suffering from growths of the kidney, only $2\frac{1}{2}$ per cent of the total, and only 2 of these were under 50 years of age.

Hæmaturia occurred in every instance of renal growth which attended the department: in 8 cases it was the presenting symptom; and in 6 the first symptom. It was usually profuse and recurrent; in only 1 instance was there a single bleeding, and this occurred at the commencement of the symptoms, a whole year before the patient attended hospital.

In 2 cases the growth was associated both with renal calculi and hydronephrosis; it was not possible to say which was the primary condition. The possibility of malignancy should be remembered in patients showing marked cachexia, profuse hæmaturia, and a big kidney with stones showing in the X-ray.

Characteristically blood was seen coming from the ureter on the affected side only. Pyelography may show either a filling defect, or sometimes may be an apparent failure because no opaque fluid enters the renal pelvis, which is blocked by growth or blood-clot.

Papilloma and Carcinoma of the Bladder.—Of all cases of hæmaturia 23 per cent were explained by bladder growth (in men 27 per cent, and in women 14 per cent). Neoplasm of the bladder is the commonest cause of hæmaturia in men, and much the commonest cause of it as the presenting symptom. Moreover, bleeding was the most marked feature of all bladder growths, and was almost always the first manifestation. In papilloma of the bladder it was the *only* symptom in 41 per cent, and the presenting symptom in 63 per cent of cases. In carcinoma the percentages were 8 and 47 respectively.

Carcinoma occurs at a later age period than papilloma, the average age for papillomata being 52 and for carcinoma 61 years; both conditions are very rare under the age of 30. Bladder growths are much less common in women than in men; for papilloma the proportions are about 1 woman to 4 men and for carcinoma 1 to 5.

Carcinoma of the bladder and prostatic enlargement are often associated, and may produce somewhat similar symptoms, so that unless cystoscopy is performed the former may masquerade as a simple prostatic hypertrophy.

Enlarged Prostate.—Ten per cent of all cases of hæmaturia in men are due to prostatic enlargement, and if carcinoma of the prostate is included, the percentage becomes nearly 13.

There were 130 cases of hæmaturia in men over 60 years of age; 39 of these were due to simple prostatic enlargement (30 per cent), and 42 were due to carcinoma of the bladder (32 per cent), the percentage for the two conditions being about the same. Of all cases of hæmaturia in men over 60, 84 per cent are caused by the following conditions:—

Simple prostatic enlargement ..	39 cases	30 per cent	} 37 per cent
Carcinoma of the prostate ..	9 „	7 „	
Papilloma of the bladder ..	15 „	11 „	} 43 „
Carcinoma of the bladder ..	42 „	32 „	
Stricture of urethra (plus complications)	5 „	4 „	

The hæmorrhage may be associated with the usual signs and symptoms of prostatic enlargement, or it may be almost the only symptom. The amount of blood varies greatly: there may be a few streaks only, or the urine may be a deep red, and clot retention may result.

Cystoscopy shows that the bleeding is usually due either to engorged veins running over the surface of the prostate or to a very congested mucosa overlying the gland. Bleeding in the former case tends to be more profuse, in attacks; and in the latter case the hæmorrhage is less and may merely cause streaks of blood in the urine. Characteristically the bleeding occurs especially at the end of the act of micturition, but this is by no means invariable.

Blood in the urine may result from the complications arising from prostatic enlargement; 8 cases were complicated by vesical calculi, 2 by prostatic calculi, 2 by acute prostatitis, and 2 by cystitis.

Carcinoma of the Prostate.—The hæmaturia in these cases conformed very much to that found with the simple enlargement. There were 15 cases, 2 were complicated by the presence of vesical calculi. The average age of the cases of carcinoma of the prostate was practically the same as that of the cases of simple enlargement.

Renal Calculi (59 cases: 37 men, 22 women).—The maximal incidence was from the age of 20 to 50 in men, and from 40 to 60 in women, but no decade was exempt.

The great majority (46 of 59 cases) had hæmaturia falling under the heading of *Group B*; it was usually associated with renal pain and conformed to text-book description. Occasionally, however, bleeding was the first symptom (3 men and 2 women), and in others (1 man and 3 women) it was the only symptom. In addition there were 4 cases in which the bleeding was unexplained at the time but later proved to be due to calculi. These patients had no pain, and the original X-ray examination was negative.

The severity of bleeding varies considerably; it may be slight or so severe as to cause anæmia. One patient came to hospital with bleeding which was

believed to be due to an injury; he had slipped and hurt his side. A routine X-ray demonstrated the cause of the 'injury,' which was a large renal calculus that had remained latent up till that time. The oft-repeated advice to X-ray accident cases again proves useful.

Ureteric Calculi (25 cases: 22 men, 3 women).—Bleeding was usually associated with and overshadowed by pain; it was, however, the presenting symptom in one case and the first symptom in another.

Vesical Calculi (20 cases: 16 men, 4 women. Average age: men 44, women 35).—Hæmaturia was usually only slight, though it may occur on several occasions, especially at the end of micturition. In 3 cases only was it the presenting symptom, all 3 being men of somewhat advanced years.

Urethral Calculus.—There was only 1 case of urethral calculus; it was a migratory calculus, which had formed in the kidney, and caused bleeding when it became impacted in the urethra.

Gravel.—An infant aged 1 year and 9 months had hæmaturia associated with pain and frequency produced by the presence of gravel. The attack was not repeated and the child was well three years later.

Tuberculosis (38 cases: 25 men, 13 women. Average age: men 30, women 36).—Five per cent of all cases of hæmaturia were due to tuberculous infection. Amongst the 2000 patients who attended the Genito-urinary Department, 77 had tuberculosis of the genital or urinary tract. Of these 38, or almost 50 per cent, had hæmaturia.

Although bleeding was usually associated with painful frequent micturition, occasionally it was the presenting symptom, and in several others the first symptom noticed was blood in the urine. Bleeding from a tuberculous kidney may precede bladder symptoms by months or even a year, the patients having no symptoms in the interval, and this should always be remembered especially when dealing with young adults.

Hæmorrhage may occur either from the kidney or bladder; a tuberculous bladder is frequently contracted, very intolerant, and bleeds readily if over-distended, so making cystoscopy difficult, and any overfilling causes bleeding which obscures the view.

Nephritis (14 cases).—These must be regarded as stray examples of nephritis, showing how careful the urologist must be to exclude patients who are usually treated by a physician. The number is not a true index of the frequency of hæmaturia due to nephritis, because most cases of this condition were treated in the Medical Department. Cystoscopy usually showed nothing abnormal; occasionally blood was seen coming from one ureteric orifice, or characteristically from both.

If the routine examination of a case of hæmaturia gives a negative result, the investigation is not complete until a full medical examination has been made, including examination of the blood and fundi and estimation of the blood-pressure.

Pyelitis (58 cases: 21 men, 37 women. Average age: men 28, women 32).—The condition occurs especially during the child-bearing age in women, and in men at about the same age (20 to 40 years). There was no case over 60 years of age.

Hæmaturia due to pyelitis is not uncommon. Sometimes a profuse

bleeding attack may occur at the beginning of the trouble, the attack may recur, or small amounts of blood may be passed at intervals.

In quite a number of cases hæmorrhage was the presenting symptom—7 men and 7 women. Bleeding is often profuse, and may be almost the only symptom, pain and frequency being slight or absent. It seems that the passage of blood in the urine may give rise to some frequency which ceases with the hæmaturia. On examination of the urine pus was present in small amount, often only detectable with the microscope, but sometimes quite obvious. Colon bacilli were usually found. Rarely, there may be simply leucocytes in a sterile urine. There are various possible sources of error, such as the accidental presence of antiseptics in the sterile specimen and mistakes in technique; nevertheless these cases appear to merge into the group of 'unexplained' hæmaturia.

Cystoscopy in cases of pyelitis shows anything from a marked cystitis (in which case it is classified as pyelo-cystitis) to a normal bladder and ureteric orifices. The negative findings may be due to the fact that the inflammation has subsided, leaving nothing more than some injection of the vessels. The typical picture shows a red œdematous ureteric orifice, with a varying amount of inflammation of the bladder wall around. Often the findings may be simply œdema of one or both ureteric orifices, with congestion of the base of the bladder (trigonitis), or petechial hæmorrhages may be seen. Blood or turbid urine may be seen coming from one or both ureteric orifices.

Pyelo-cystitis.—The age incidence is the same as and the history similar to that given in cases of pyelitis, except that the bladder symptoms are more marked, though in this group there were 7 men and 2 women who came up with hæmaturia as the presenting symptom. The groups of pyelitis and pyelo-cystitis naturally merge into each other.

Cystoscopic appearances show anything from an intense general cystitis, usually associated with a most intolerant bladder, to a mild basal cystitis, often associated with submucous hæmorrhages similar to those seen in purpura of the urinary tract.

[CONCLUSIONS.—Evidently a number of different types of case are classed as pyelitis.

1. There is the typical text-book type of case, seen commonly in the medical wards, acutely ill, with high fever and perhaps a rigor at the onset, marked pyuria, and acute tenderness over the kidney.

2. A milder type attends the out-patient department: the symptoms are mostly of renal character with lumbar pain and frequent micturition; constitutional disturbances are considerably less marked.

3. There is an even milder type in which hæmaturia is marked, pyuria almost absent, and malaise very slight. Indeed, the condition seems to be little more than a renal epistaxis, and it is difficult to decide whether to class the case as one of pyelitis or as one of unexplained hæmaturia. Some of these cases may be due to purpura of the kidney.

These marked differences probably depend largely upon the virulence of the infection and the amount of renal tissue involved.]

Cystitis (68 cases: 30 men, 38 women. The average age for both sexes was 39, but cases occurred at all ages).—A variety of cases fall under the

heading of cystitis, the causes of which are numerous; in many cases the exact etiology is difficult to ascertain, but it is probable that many of them should be classed under the heading of pyelo-cystitis.

Nine cases were considered to be due to cystocele, a common cause of cystitis in women, and one that is often overlooked. Cystocele was present in other cases too, but the connection with the cystitis was not so certain.

Six cases were associated with neuropathic bladder with secondary cystitis (4 men and 2 women); there was a definite spinal-cord lesion in 5 of these. One case must be classed as an ulcer of the bladder; no cause for it could be found.

The hæmaturia characteristically occurred in small amounts, blood being passed at the end of the act of micturition. Often there were shreds of blood only. Sometimes hæmorrhage was profuse. Cystoscopy showed an intense cystitis with engorged vessels and red mucosa, the comparative freedom from pain and frequency being sometimes quite surprising. Pain and frequency usually figure prominently in cystitis, and it is rather surprising that 14 of these cases came to hospital with hæmaturia as the presenting symptom.

Prostato-cystitis and Prostatitis (9 cases).—Bleeding was not profuse; it often occurred at the end of micturition.

Cystoscopy was performed in all but two cases, and showed marked congestion and dilatation of the veins over the prostate, or a basal cystitis; this evidently accounted for the bleeding. In acute cases of prostatitis cystoscopy is contra-indicated.

Urethritis (3 cases: 1 man, 2 women).—Each case was associated with a purulent urethral discharge, most probably of gonococcal origin. The hæmorrhage was at the end of the act of micturition, and was apparently due to associated cystitis. Cases of venereal disease were not treated in the department, and must be regarded as stray cases.

Pyonephrosis.—One case of old-standing pyelitis, and later pyonephrosis, with adhesions kinking the ureter, was associated with very slight hæmorrhage.

Bilharzia.—One case of hæmaturia as a presenting symptom occurred in a boy of 18 years who had previously suffered from bilharzia. A slight injury to the abdomen produced an attack of hæmorrhage.

Polypi round Internal Urinary Meatus.—A woman of 52, with hæmaturia as the presenting symptom, was found to have polypi around the internal urinary meatus; these were most probably inflammatory in nature.

Stricture of the Urethra (23 cases, all men. Average age 47—youngest 21, oldest 72).—Hæmaturia, which was usually only slight and occasional, occurred only in complicated cases and as a result of infection supervening on the stricture—prostatitis, cystitis, calculus formation, and so on.

In some cases there was a long-standing history suggesting stricture, with recent symptoms of cystitis; in others the patient apparently had an acute cystitis without any obstructive symptoms, and the underlying cause of the trouble was only discovered on attempted cystoscopy and confirmed by urethroscopy.

Diverticulum of Bladder (3 cases, all men. Average age 42 years).—Although the diverticula were all congenital, and the youngest patient 31

years old, the longest history was of only a couple of months' duration. The symptoms were produced by complications—cystitis or calculus formation.

Trauma.—All the cases of hæmaturia resulting from trauma occurred in men; 6 patients received injuries in the kidney region, causing bleeding from the kidney; 3 received blows on the perineum and so caused bleeding from the urethra; 1 case was caused by straining during coitus.

Foreign Body in the Bladder.—There were 3 cases of this condition, all in young people; none of them gave any history of the presence of the foreign body until it had been discovered on cystoscopy. The history in each case was one of a subacute cystitis, with considerable bleeding, produced either by the foreign body or by resulting infection. All the cases of foreign bodies in the bladder which attended the department had hæmaturia. The foreign bodies were a pen-holder and a piece of tallow candle, found in the bladders of men of 25 and 19 years of age respectively, and a slate pencil in the bladder of a woman of 19; the last case also had a perivesical abscess.

Hæmaturia from Varicose Veins of Bladder (4 cases: 3 men, 1 woman. Average age: men 53, women 29).—Hæmaturia was the presenting symptom in each case. On cystoscopy the tortuous varicose veins were seen over the base of the bladder. Great caution must be taken in diagnosing these cases, for it is easy to attribute bleeding to an engorged varicose vein of the bladder when it has actually come from a kidney, and the bleeding has ceased before cystoscopy is performed.

In the case of the woman the bleeding occurred especially during pregnancy; she was quite well otherwise, but during two pregnancies she attended with hæmaturia, which was found to come from a large tortuous vein running across the trigone just behind the internal urinary meatus.

One man, who first attended at the age of 66, had such severe bleeding that it was necessary on two occasions to open the bladder and deal with the bleeding point.

Congenital Cystic Kidney (6 cases: 1 man, 5 women. Average age: men 53, women 31).—There was a family history of the complaint in 2 cases. Hæmaturia is a characteristic symptom of this condition and occurred in every case which attended the department. The bleeding occurs intermittently, in attacks, and may be quite profuse; it is often associated with a dull ache in the loins; pyelitis is not uncommon as a complication.

Purpura of the Urinary Tract (6 cases: 2 men, 4 women. Average age: men 27, women 36).—In every case hæmaturia was the presenting symptom; pain was present in 3 out of the 6 cases, but in no instance was it severe. Each gave a history of quite profuse bleeding, lasting from one to eight days; sometimes the attack had recurred.

Cystoscopy showed purpuric spots scattered over the whole bladder; no spots were found elsewhere. *B. coli* was cultured in the urine of 3 of the women; in no case was pyuria present. One man, aged 62, had chronic heart disease, with auricular fibrillation.

Of the 6 cases which could be traced, all remained quite well when inquiry was made from six months to six and a half years after the attacks of hæmaturia. The prognosis is therefore good.

Hydronephrosis.—Of the 3 cases of hydronephrosis (average age 36), 2 gave histories of renal pain with attacks of hæmaturia, and 1 a history of hæmaturia only; there were no other symptoms except an occasional feeling of discomfort in the right side of the abdomen.

Pyuria was present in all the cases, and all were operated on, the clinical findings being confirmed. The bleeding was probably due to congestion and slight infection.

Rena Mobilis.—There was one case of rena mobilis producing typical renal colic which was followed by hæmaturia on one occasion, and another case which was associated with trigonitis and slight hæmaturia.

Varicose Veins of the Urethra.—There was an interesting case of a man with varicose veins of the urethra. He first attended at the age of 14 with a two weeks' history of bleeding from the urethra independent of micturition; there were no other symptoms. Urethroscopy showed a small cluster of varicose veins extending for $\frac{3}{4}$ in. along the lateral wall of the urethra, and situated $1\frac{1}{2}$ in. from the external urinary meatus. No other abnormality was found. He returned some years later with a recurrence of the trouble; the veins were then cauterized with success.

Vesico-vaginal Fistula.—In one case a vesico-vaginal fistula followed hysterectomy; this was complicated by cystitis and vesical calculus, and hæmaturia resulted.

Blood Dyscrasia (? Hæmophilia).—There was an interesting case of a man of 29 who first attended with a five days' history of hæmaturia followed by an attack of left renal colic (evidently clot colic). The family history was that the grandfather was a bleeder. Both parents were healthy and well. The patient had three brothers; two were well, one had been in hospital twice with hæmorrhage from the kidneys, but no operation had been performed. He had three sisters, all alive and well. The patient himself had had four teeth extracted on different occasions, and the sockets had to be plugged for bleeding each time.

On investigation there were no abnormal physical signs. The urine contained albumin and blood. Cystoscopy showed that blood was coming from the left ureteric orifice. A pyelogram of the left kidney was normal. A blood-count and platelet-count were also normal. The blood coagulation time was 3 minutes 30 seconds (normal, 2 minutes 45 seconds).

Eighteen months later the bleeding recurred for five days. Investigations were negative; the bleeding had ceased when cystoscopy was performed. The blood coagulation time was the same as that found on the first occasion. For the last two years he has remained perfectly well.

UNEXPLAINED HÆMATURIA.

There were 66 cases of hæmaturia the cause of which was not explained by investigation (40 men and 26 women). Letters were written to each patient to discover the after-history; 17 could not be traced or did not reply (10 men and 7 women). There are, therefore, 49 cases to be considered. Of these, 4 (8 per cent) proved later to be due to calculi; 27 (55 per cent) had no more trouble of any kind; and 6 (12 per cent) had no more hæmaturia, but slight

other symptoms. Adding these two groups together, 67 per cent remained well. Four (8 per cent) died, the cause of death having no definite connection with the bleeding except perhaps in one instance. Eight (16 per cent) had recurrence of the bleeding; however, all except one of these remained fairly well and none of them attended for further investigation.

The groups are now considered seriatim :—

Inquiry Made: Hæmaturia Explained.—All these 4 cases were due to renal calculi. Hæmaturia had been the presenting and almost the only symptom when they attended hospital. X-ray examination had been negative. Two patients passed stones later; one had a stone removed at operation, and one attended hospital as a result of the inquiry, when further X-ray examination demonstrated the calculus six and a half years after her first attendance.

Cases of Unexplained Hæmaturia which Remained Perfectly Well.—There were 27 patients (15 men and 12 women) in whom the cause of bleeding had not been discovered and who remained perfectly well. The average age of the men was 32 and that of the women 41 years. The cases fell into the following groups :—

Hæmaturia as the presenting symptom	..	13 men	6 women
„ as an important symptom	..	2 „	5 „
„ as an incident	..	0 „	1 „

All were well, and hæmaturia had not recurred when inquiry was made at an interval of six months to seven and a half years (average time three years) after their first attendance at hospital. The group probably represents a variety of conditions.

Of the 15 men, bacteria were cultured in an otherwise clear urine in 5 cases, and infection may have been responsible. Casts were found in the urine of another. One had slight oxaluria, one a positive Wassermann reaction, 1 refused adequate investigation, and in 6 there was nothing abnormal to be found.

Of the 12 women, bacteria were cultured in an otherwise clear urine in 4 cases, 1 was probably a case of purpura, 1 had a movable kidney, 1 had a few casts in the urine, and in 5 nothing abnormal was found.

Inquiry Made: No More Hæmaturia, but Slight other Symptoms.—There were 6 cases of unexplained hæmaturia who remained well except for slight discomfort (5 men and 1 woman). They represent a variety of conditions, and little importance can be attached to the cases. One case was probably a mild infection, 1 suffered from nocturnal enuresis, 3 had slight back-ache but were otherwise well, and 1 had some pain following exploratory nephrotomy.

Inquiry Made: Hæmaturia Recurred.—The 8 cases in which hæmaturia recurred are as follows :—

Case 1.—Male, aged 14 when he first attended hospital. On several occasions he had attacks of bleeding, which were seen on one occasion to be coming from the left kidney. Investigations were otherwise negative, and he was well eleven years later—so well, in fact, that he did not wish to attend for any further investigations, although he continued to have hæmaturia about once a year.

Case 2.—Man, aged 28, with symptomless hæmaturia which had recurred on two occasions; he was well two years after attending hospital.

Case 3.—Man, aged 31, who attended with symptomless hæmaturia which recurred two years later. He had no other trouble, and was well three years after the last bleeding.

Case 4.—Man, aged 49. Attended with symptomless hæmaturia; investigations negative, except that he had syphilis. During the next three and a half years he had two recurrences of the bleeding and became blind. He was unable to attend for further investigation.

Case 5.—Man, aged 49. Symptomless hæmaturia, except for one attack of left renal pain (probably clot colic). Investigations showed that on one occasion blood was coming from the left ureter. Pyelograms showed slight bilateral hydronephrosis, and streptococci were cultured in the urine obtained from the left kidney. He was well four years later except for occasional recurrence of the bleeding. The case was probably one of chronic streptococcal infection.

Case 6.—Man of 42, with symptomless hæmaturia; investigations negative. Doubtful occasional recurrence of the bleeding during the following year, otherwise he was well.

Case 7.—Woman, aged 38. Exploratory nephrotomy and nephropexy had been performed five years before she attended the department. She continued to complain of left renal pain and occasional hæmaturia. Investigations inconclusive; pyelogram normal. On inquiry another five years later (i.e., ten years after operation) she still suffered from renal pain and occasional hæmaturia but was otherwise well; urine offensive. Diagnosis: suggestive of secondary infection.

Case 8.—Woman, aged 54. Attended with painless hæmaturia as the presenting symptom; some frequency; blood-stained urine seen coming from left kidney on one occasion. Pyelogram normal. Attended for two years, with occasional bouts of slight hæmaturia (smoky urine); no bleeding for past year: otherwise well.

No Reply Received to Letters of Inquiry.—There were 17 cases (10 men and 7 women) in which the hæmaturia was not definitely explained. These patients could not be traced, so little benefit will result from a detailed consideration of them, but mention will be made of the probable diagnosis.

Ten men (average age 29 years): 1 probably cystitis; 1 probably varicose veins of bladder; 1 probably oxaluria; 3 probably pyelitis; 1 hæmaturia associated with extreme changes of temperature; 3 nothing abnormal found.

Seven women (average age 29 years): 2 probably cystitis; 1 probably *B. coli* infection; 1 probably varicose veins of bladder; 2 associated with renal colic; 1 probably renal calculus.

Inquiry Made: Had Died.—There were 4 cases of unexplained hæmaturia (2 men and 2 women) who had died. Probably in only one case was the cause of death due to the trouble for which they attended hospital.

Case 1.—Man, aged 50. Hæmaturia perhaps due to slight prostatic enlargement. Remained perfectly well whilst he was kept under observation at hospital for fifteen months. Died of acute lung condition.

Case 2.—Man, aged 54. Symptomless hæmaturia. Nothing abnormal found except slight prostatic enlargement. Died of carcinoma of the prostate eight years later.

Case 3.—Woman, aged 38. Hæmaturia and frequency associated with pregnancy on two occasions. Died of pneumonia.

Case 4.—Woman, aged 64. Symptomless hæmaturia for four weeks. Investigations negative. Died of 'stroke' two years later.

DISCUSSION AND REVIEW OF THE LITERATURE.

The findings given in this paper appear to show that very few cases of hæmaturia are unsatisfactorily assessed in a modern urological department: though the actual cause of the bleeding was not always discovered, those cases which were unexplained did not suffer in consequence. Pure uric acid calculi which are not opaque to the X rays appeared to be a cause of missed diagnoses (these, however, can be shown up by pyelography, the stone appearing as a radio-translucent area around the opaque medium).

As stones may form around a nucleus of blood-clot, it may be that these calculi were not present when the patient attended hospital, but formed afterwards on a nucleus of blood-clot or fibrin which had not been voided.

The importance of hæmaturia as a symptom still needs to be stressed: all cases should be investigated and if possible explained. Too often patients attend hospital with a long-standing history of repeated attacks of bleeding; if pain is absent, both patient and doctor are often satisfied to await further developments rather than submit to an investigation in the early stages, when results of treatment are much more satisfactory.

The dangers of hæmaturia *per se* are slight; the loss of blood is rarely so severe as to endanger the patient's life, and it does not cause the profound collapse which is found with a severe hæmatemesis or hæmoptysis, though considerable anæmia, of course, results from persistent bleeding.

The symptom is a valuable one, for it immediately directs attention to the urinary tract, and is therefore of great diagnostic importance. About one-third of the patients attending the Genito-urinary Department gave histories of hæmaturia. Though it focuses attention on the urinary tract, the primary lesion is not necessarily situated there; one case seen recently first attended with hæmaturia which was subsequently explained by a sub-acute infective endocarditis. Not uncommonly the bleeding arises from a complication which has occurred as the result of a lesion in the urinary tract: for instance, it may be due to a vesical calculus complicating prostatic enlargement, or to cystitis following on a stricture of the urethra. In women supposed hæmaturia may in reality come from the genital tract, and this point occasionally needs attention.

It has been noticeable throughout this investigation that despite the fact that chronic inflammatory conditions are so much more common in women than in men, neoplasm is comparatively rare: this observation is at variance with the oft-repeated assertion that chronic inflammation predisposes to carcinoma, and it seems that we shall have to look elsewhere for the explanation of this phenomenon in the urinary tract.

During recent years Kretschmer,¹ of Chicago, has reported the most complete series of cases of hæmaturia. He reports 933 consecutive cases, 860 of which were diagnosed. The general similarity between the two series

is quite marked, the chief differences being that neoplastic conditions and tuberculosis are more common in Kretschmer's series, whereas other inflammatory conditions are less common.

Mackenzie² summarizes a series of 821 consecutive cases of hæmaturia which occurred amongst the 3800 patients who attended the Department of Urology at the Royal Victoria Hospital, Montreal. He finds that "excluding the urethra 536 cases out of 761, that is. over 70 per cent, were caused by calculi, tuberculosis, cancer, or surgical lesions of the kidney."

Van Duzen³ reported a series of 500 cases; the source of the bleeding was: urethra 38 cases, prostate 59 cases, bladder 82 cases, ureter 75 cases, and kidney 172 cases.

Cases of unexplained hæmaturia occur at all ages and in both sexes. The hæmaturia is especially likely to be unexplained when it is the presenting symptom or the only symptom and when the case is seen after the bleeding has ceased. If nothing abnormal can be found on examination, the prognosis is good.

If the bleeding has been localized to one kidney, and nothing abnormal is found on thorough examination and repeated pyelograms are normal, conservative treatment should be adopted unless bleeding endangers the life of the patient or produces a grave anæmia. If exploratory nephrotomy is performed, most probably nothing abnormal will be found, and the surgeon may remove a normal kidney thinking that he is dealing with an early malignant neoplasm. Investigations indicate that this is most improbable.

The literature on essential hæmaturia must be referred to in connection with these unexplained cases. Various explanations of it have been given; Braasch⁴ says: "I am under the impression that it would be difficult to assign any one cause to the variety of conditions which are grouped under the term essential hæmaturia", and the findings of this investigation support his view.

Some urologists believe that the diagnosis of essential hæmaturia serves as a cloak for ignorance of the exact condition, and think that the bleeding is in reality the early manifestation of some serious disease such as nephritis, carcinoma, or tuberculosis.

Bumpus⁵ reviews 155 cases of essential hæmaturia five to twenty years after the diagnosis had been made at the Mayo Clinic: only 6 of these reported the development of any definite renal disease. In 3 calculi developed, and in 3 others nephrectomy was performed for unknown reasons (? for persistent bleeding): 21 had died, 105 patients were reported in good health, 44 in fair health, and 6 in poor health. He concludes that "we may dismiss the idea that the hæmaturia was caused by early malignant growth or any other serious disease in its incipency". Levy⁶ came to a similar conclusion, and the same results are shown in this thesis.

The various conditions which are believed to be the cause of essential hæmaturia are well discussed by Bumpus. They may be summarized thus:—

1. *Nephritis*.—Bumpus found no confirmation of this explanation in the follow-up of his 155 cases; the present investigation corroborates his view.

2. *Chronic Passive Congestion*.—This has been suggested as a cause of the bleeding (Spitzer,⁷ Rathbun⁸).

3. *Dilated Veins or Varix Papillæ*.—These, with or without associated inflammation, appear to be definite causes in some cases; so called papillitis or angioma of the papillæ.

4. *Infection*.—Focal infection may sometimes be responsible; septic tonsils or infected teeth are often found, and the removal of these appears to clear up a number of cases. Kidd⁹ attaches much importance to the removal of septic foci in patients with purpura of the urinary tract.

5. *Blood Dyscrasia*.—This probably accounts for a few cases. Conner and Bumpus¹⁰ paid particular attention to this theory in a study of 33 cases; they found little evidence that the disease was a localized purpura hæmorrhagica except for the low platelet-count, which averaged 136,000 for the group, about half the normal. They suggest that the deficiency in platelets may have a bearing in some cases. One case in the present series appears to be an example of a blood dyscrasia.

It seems that there are numerous causes of so-called essential hæmaturia; the lesion is a minor local lesion of the kidney of various kinds; indirect treatment is usually all that is required. Very occasionally if the bleeding is so severe as to endanger the patient's life, operation is indicated.

'Essential hæmaturia' is an unsatisfactory name, and it has been said should be used "only in the quietness of one's study, or with very intimate friends, but never to an intelligent patient." The time-honoured name of 'renal epistaxis' given by Sir William Gull, seems to be more satisfactory, and more accurate, since it does not lay unnecessary stress upon what is, after all, only a minor local lesion of the kidney.

SUMMARY OF CONCLUSIONS.

1. Papilloma and carcinoma of the bladder are the commonest causes of hæmaturia in men.

2. Inflammatory conditions of the urinary tract are the commonest causes of hæmaturia in women.

3. Hæmaturia as a presenting symptom: In men there is a 50 per cent chance that it is due to a papilloma or carcinoma of the bladder. In women the cause is as likely to be inflammatory as neoplastic.

4. Hæmaturia as an only symptom (65 cases): In men there is a two to one chance that it is due to papilloma or carcinoma of the bladder. In women (10 cases) the commonest cause was renal calculus.

5. Over the age of 50 the prostate is a common cause of hæmaturia in men.

6. In 130 cases of hæmaturia in men over 60 years, 43 per cent were suffering from papilloma or carcinoma of the bladder, and 37 per cent from simple or malignant enlargement of the prostate.

7. Cases of carcinoma of the bladder are not infrequently associated with prostatic enlargement; the symptoms are in some cases rather similar; unless cystoscopy is performed a case may for a time be considered to be one of prostatic enlargement and so valuable time be lost.

8. The common causes of hæmaturia in the different decades of life are shown on a graph. The conclusion is that hæmaturia is rare in the first two

decades. In the second and third decades inflammatory conditions and calculi are the commonest causes in both sexes. Neoplasm becomes the commonest cause of hæmaturia in men in the fifth decade of life, and in both sexes in the sixth decade of life.

9. Carcinoma of the kidney is rare; it was the cause of only $2\frac{1}{2}$ per cent of all the cases of hæmaturia (men 3 per cent, women less than $\frac{1}{2}$ per cent).

10. Renal calculi not uncommonly cause hæmaturia which may be almost or quite painless.

11. The diagnosis of hæmaturia due to varicose vesical veins must be made with very great caution, unless the bleeding is actually seen to be coming from a varix.

12. Cases of moderate and well-marked hæmaturia should be cystoscoped during the attack of bleeding.

13. Cases suggestive of carcinoma vesicæ with very foul urine containing much blood are usually cystoscoped more satisfactorily after rest in bed for a few days and treatment to the bladder, otherwise nothing can be seen through the dirty medium.

14. Of the 66 cases of unexplained hæmaturia 49 were traced, and the after-history discovered, and of these:—

Four cases (8 per cent) reported that calculi had been either passed or removed.

Thirty-three (67 per cent) were almost or quite well, no more bleeding having occurred.

Eight (16 per cent) reported recurrence of bleeding on one or more occasions. All were well or only slightly inconvenienced, except one man who had syphilis and had become blind.

Four (8 per cent) had died, two of acute lung conditions quite unconnected with the hæmaturia, one of carcinoma of the prostate eight years after his attendance at hospital, and one of stroke.

The most difficult cases to diagnose are those giving few symptoms other than the hæmaturia which has ceased by the time the patient attends hospital. Cases of unexplained hæmaturia fall into several different categories; the prognosis is good if nothing abnormal can be found on full urological investigation.

I am greatly indebted to Mr. Hugh Lett both for permission to investigate these cases and for his kind advice; also to Dr. Joules for helpful criticism.

REFERENCES.

- ¹ KRETSCHEMER, H. L., *Surg. Gynecol. and Obst.*, 1925, xl, 683.
- ² MACKENZIE, D. W., *Ibid*, 1924, xxxix, 155.
- ³ VAN DUZEN, R. E., *Texas State Jour. Med.*, 1928, xxiii, 577.
- ⁴ BRAASCH, W. F., *Trans. Amer. Assoc. Gen.-urin. Surg.*, 1926, xix, 276.
- ⁵ BUMPUS, H. C., *Jour. Amer. Med. Assoc.*, 1928, xc, 593.
- ⁶ LEVY, C. S., *Surg. Gynecol. and Obst.*, 1922, xxxiv, 22.
- ⁷ SPITZER, W. M., *Ibid*, 1914, lxiii, 2110.
- ⁸ RATHBUN, N. P., *Trans. Amer. Assoc. Gen.-urin. Surg.*, 1926, xix, 253.
- ⁹ KIDD, F., *Proc. Roy. Soc. Med.*, 1928, xxi, 19.
- ¹⁰ CONNER, H. M., and BUMPUS, H. C., *Amer. Jour. Med. Sci.*, 1927, Feb., 176.

A CASE OF RENAL AND URETERIC CALCULI OF UNUSUAL SIZE. REPEATED FORMATION OF CALCULI.

By EDGAR FRESHMAN,

SURGICAL FIRST ASSISTANT AND REGISTRAR, LONDON HOSPITAL.

THE following case is an interesting example of 'continuous' stone formation in the urinary tract, and illustrates the enormous size that may be obtained by calculi before giving rise to symptoms severe enough to induce the patient to seek relief:—

FIRST ADMISSION.—A man of 22, by occupation a clerk, was admitted to the London Hospital in June, 1923, in a typical attack of left renal colic, which lasted for about twelve hours. He had had no previous attacks, but gave a history of having had hæmaturia on two occasions during the past twelve months. The urine contained pus and blood. Two days later he had a sudden stoppage of the flow during micturition, accompanied by severe pain in the penis. A tender mass was palpable in the urethra, one inch from the external meatus, and an attempt at catheterization confirmed the presence of a calculus. An anæsthetic was administered for the purpose of urethroscopy and removal of the calculus, but this was at once passed, in front of a stream of urine; it was the size and shape of a date-stone.

Six days later another stone became impacted at the same spot, again causing acute retention of urine. The patient was anæsthetized, and again the retention was relieved by the immediate passage of the calculus, which was about half the size of the first one.

X-ray examination revealed a group of shadows towards the lower pole of the left kidney; the right kidney was clear. On July 12, 1923, the left kidney was explored; a moderate hydronephrosis was found, most marked towards the lower pole, and a number of small stones were removed. The renal pelvis was drained through an incision in the cortex. A urinary fistula persisted, and on exploration a little pus was evacuated from the kidney, and the ureter proved to be patent and unobstructed throughout its length. The fistula healed rapidly, and the patient was discharged as fit on August 25, 1923.

SECOND ADMISSION.—In November of the same year he was re-admitted. He stated that for two months he had had pain in the perineum and at the tip of the penis at the end of micturition, and momentary painful stoppage of the stream in the middle of the act. He had had no hæmaturia, but his urine had been 'brick-red with gravel'. He was always most comfortable when the bladder was full. Radiography revealed one vesical and one renal calculus, this time on the right side, lying in the body of the kidney. After a confirmatory cystoscopy, the vesical calculus was crushed and removed, with a view to subsequent removal of the one in the kidney.

A few days later the patient had an attack of right renal colic—the first caused by the hitherto 'quiescent' right renal stone, and a further X-ray examination showed that it had moved into the pelvis of the kidney. A right nephrotomy was performed, and a calcium carbonate stone, 2.5 cm. long and 1.5 cm. broad, was removed from the moderately dilated pelvis, and a smaller one, whose shadow had evidently been hidden by that of the larger, from a dilated major calyx. The kidney was drained, and the patient was discharged five weeks later.

THIRD ADMISSION.—The urine remained rather dirty, but he was free from symptoms until a short time previous to his re-admission to hospital in

August, 1924, on account of his having had several further attacks of right renal colic during the preceding few weeks, the last one culminating in the passage of a small calculus the day before admission. Two calculi, the larger 2.5 cm. in diameter, were seen in the *left* kidney on X-ray examination, the right side being clear. Chromo-cystoscopy showed depressed function of the left kidney, that of the right being normal. There was no pus in the ureteric catheter specimen of urine from either side; a culture of that from the left side gave a growth of *B. proteus*, but nothing from the right. A left nephrotomy was again performed, through as small an incision as possible, and two calcium carbonate stones were removed



FIG. 43.—Radiograph showing renal and ureteric calculi.

from the renal pelvis. The kidney was drained, and the patient discharged with a soundly healed wound two and a half weeks later.

He did not report again until March, 1932. He had remained perfectly well until about the middle of 1930, when he began to get attacks of aching pain in the left lumbar region, which 'passed through from back to front', but never radiated towards the groin. There were long intervals of freedom between the attacks, which invariably occurred at the end of a heavy day, and were relieved by rest, hot drinks, and hot packs. There were no other symptoms, and the urine had remained clear until November, 1931, when there was an attack of hæmaturia. Another followed in three weeks, and a

fortnight later the patient had an acute attack of left renal pain, with no radiation. The radiograph (*Fig. 43*) showed an enormous beaked and branched calculus lying in the upper part of the left kidney, with its beak in the pelvis, and two irregular stones lying below it. The left ureter contained a huge rod-shaped calculus in its middle third, and a three-sided one opposite the ischial spine. The right kidney and ureter appeared clear.

FOURTH ADMISSION.—The patient was re-admitted to hospital in April, 1932. The total urinary function was satisfactory (blood-urea 0.025 per



FIG. 44.—Calculi removed at operation. A, Renal; B, Ureteric.

cent; urea concentration 1.98 per cent, and 2.26 per cent), and the urine contained no pus. Cystoscopy showed small calculous debris lying on the trigone, especially near the left ureteric orifice, which was almost closed, and had somewhat irregular and rigid-looking margins. Its contractions were infrequent and spasmodic. Intravenous injection of indigo-carmin produced dye from the right side in five minutes, and none from the left in fifteen.

On April 5, 1932, left uretero-nephrectomy was performed. Under the anaesthetic, the large ureteric calculus was easily palpable through the abdominal wall. A gridiron incision was made in the left iliac fossa, and the left ureter exposed by stripping inwards the peritoneum, to which it was

extremely adherent. The smaller calculus was milked upwards, the ureter divided below it, a little below the level of the pelvic brim, and separated upwards as far as possible. The usual oblique kidney incision was then made in the loin, where the abdominal wall was largely replaced by scar tissue, to which the kidney was firmly adherent, with the result that the incision opened directly into a calix, liberating thin purulent urine. This incision was enlarged, and the three calculi were removed. The kidney was a mere shell of fibro-fatty tissue, very adherent to the peritoneum and splenic flexure; the ureter was rigid and fibrous and was firmly adherent to the peritoneum. The kidney and practically the whole of the ureter with its contained calculi were removed in one piece with considerable difficulty. The wound healed satisfactorily, and the patient was discharged eighteen days later.

Fig. 44 depicts the calculi removed at the last operation. As is seen in the radiograph, the large renal stone lay with its long beak in the pelvis and its branching body in the upper calices. The composition of all five was the same—namely, calcium and magnesium phosphate, with traces of calcium oxalate and carbonate. The measurements of the two largest were as follows:—

Renal calculus	..	Maximum length	7.2 cm.	}	Weight 1.45 oz.
		breadth	5.5 "		
		thickness	3.2 "		
Ureteric calculus	..	Length	7.6 cm.	}	Weight 0.86 oz.
		Maximum diameter	2.0 "		

Now, fifteen months after his last operation, the patient is in excellent health, and on X-ray examination his right kidney appears to be free of stones. Intravenous pyelography shows good excretion in five minutes, and an almost normally shaped pelvis and calices.

COMMENTARY.

The patient thus underwent six operations; on seven occasions he was relieved of a calculus or calculi under general anaesthesia, and one he voided spontaneously.

The rate at which he formed stones is worthy of note. All calculi then visible on X-ray examination were passed per urethram or removed from the left kidney in July, 1923. Four months later he was found to have two in the opposite kidney, and one in the bladder. Ten months after this, another, which according to the symptoms had also come from the right side, was passed, and two more were removed from the pelvis of the left kidney—i.e., fourteen months after nephro-lithotomy had been performed on that side. An interval of nearly six years elapsed before the onset of symptoms suggesting the presence of further calculi. None of the stones had a core of pure uric acid, which might have accounted for their existence some time before becoming opaque to X rays.

I am greatly indebted to Mr. Hugh Lett, under whose care the patient has been since he first attended hospital, for his kind permission to publish these notes on the case, and also to Dr. Vilvandré for allowing me to reproduce the radiograph.

OSTEO-CHONDRITIS DISSECANS.*

BY H. A. T. FAIRBANK, D.S.O.,

SENIOR ORTHOPÆDIC SURGEON TO KING'S COLLEGE HOSPITAL.

OSTEO-CHONDRITIS DISSECANS may be defined as a condition in which a fragment of articular cartilage and subchondral bone becomes separated, partially or completely, from typical positions at the ends of certain of the long bones.

The term 'osteochondritis dissecans' is retained purely for convenience, since I believe it has come to mean to the surgeon a definite pathological condition, though, as I shall hope to prove, the name is hardly justified by the etiology. Though usually found at definite sites of election in the joints affected, the typical lesions are occasionally seen elsewhere on the articular surfaces. It is characteristic, however, of this affection that the lesions, with few exceptions, are found in the deeper parts of a joint, where direct damage from external violence is least likely to occur. It is usually said that males are far more commonly affected than females. Phemister,¹ for instance, gives the proportion as 13 to 1, but this has not been our experience. In a small personal series of 20 cases affecting the knee-joint, the sexes were equally divided. The patients are usually adolescents or young adults, in perfect health, vigorous, and indulging in the usual strenuous pastimes of their age. The youngest patient on whom I have operated for a typical lesion in the knee-joint was a girl of 9 (*see Figs. 48, 49.*) Phemister reports a case in a girl of 12.

Trauma is a common and marked etiological factor, but a history of it is by no means always obtained. The joint most commonly affected is the knee, but similar lesions have been described in the elbow-, ankle-, and hip-joints, and even in a metatarsal head (Richards²). Bilateral lesions have been reported by several writers. I am indebted to Mr. Rowley Bristow for slides of bilateral lesions in the knees of a boy, with symptoms only in one. Operation on the painful knee revealed an area of cartilage outlined by a thin yellow line, along part of which an incision was made. As the fragment of bone beneath was firm, nothing more was done. Two months later there were no signs of disability in the joint. Somner reports a case with both knees and one elbow affected.

The typical situations for the lesions in the various joints are as follows: in the knee, the inner condyle, close to the intercondylar notch; in the elbow, the capitellum; in the ankle, the trochlear surface of the astragalus; in the hip, the highest point of the femoral head. Cases with the external condyle

* An abstract of this paper was read at the Centenary Meeting of the British Medical Association (Orthopædic Section).

of the femur affected have been reported by Balensweig,³ Delchef,⁴ and Heine,⁵ and bilateral lesions of this condyle by Niessen.⁶ We have met with lesions of the external condyle and also of the patella. If the fragment is not displaced, the symptoms are those of a chronic joint trouble of mild character, such as vague discomfort with persistent or recurrent fluid—both aggravated by violent exercise—weakness, loss of confidence in the joint, and so on.



FIG. 45.—Radiogram showing typical lesion in knee-joint. Boy, aged 14. Cut foot: laid up two weeks. On walking again noticed knee bent and swollen. Pain on extension.

On examination of the *knee*, for instance, nothing may be found but the signs of fluid and wasting of the quadriceps, and possibly some limitation of movement. Hellström⁷ says pain on extension is common. The only special sign, first described by Axhausen,⁸ I believe, and certainly present in some cases, is tenderness on deep pressure over the site of the lesion when the knee is flexed. If the fragment has become completely displaced, the symptoms will be those of a loose body. Symptoms may, however, be absent, as, for instance, in a bilateral case discovered by routine X-ray examination for symptoms confined to one knee. The radiographic picture is characteristic and determines the diagnosis. In the lateral portion of the

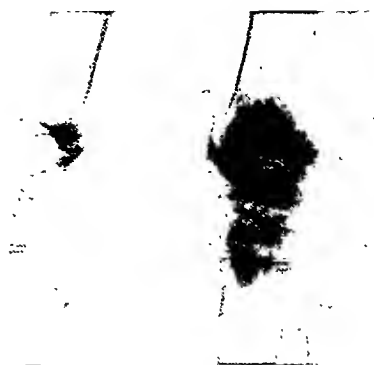
inner condyle is seen a fragment, apparently loose and lying in a depression of appropriate size and shape (Fig. 45). As a rule the fragment is single, but it may be multiple—a nest of two or three pieces (Jansson⁹) (Figs. 46, 47).



FIGS. 46, 47.—Multiple fragments—'nest of pieces'—lying in depression seen at typical site on inner condyle. Male, aged 27. Blow on knee five weeks previously. Severe swelling.

It may lie snugly in its bed, being marked off by a clear line from the rest of the bone, or it may appear to be hanging by a hinge, the amount of displacement in some cases varying with the position of the joint at the

FIGS. 48, 49.—Skiagram showing replacement of hinged fragment by full extension of joint. Girl, aged 9. Six months' history of pain and swelling. No definite trauma.



FIGS. 50, 51.—Skiagram showing crater in inner condyle and completely detached fragment in suprapatellar pouch. Operation confirmed the above. Male, aged 19. Footballer, but no definite injury. Swelling for three months before knee became locked momentarily.



FIGS. 52, 53.—Knee showing typical lesion with partial detachment of fragment. Boy, aged 16. Long history with several injuries.

time of the examination (*Figs. 48, 49*). On the other hand, the condyle may show a pond-like depression, the fragment, completely displaced, lying in the intercondylar notch or elsewhere in the joint (*Figs. 50, 51*). Frieberg¹⁰ says that in one case the 'dense nucleus' increased greatly in size in the course of a year before operation. At this stage there are usually no X-ray signs of arthritic changes.

The actual condition of the lesion as revealed at operation varies. Taking the knee as an example, in a case with a typical radiogram (*Figs. 52, 53*) we may expect to find the following. Fluid in excess is commonly present, and this may be stained with blood or altered blood-pigment. The site of the fragment is incompletely outlined by a fissure in the cartilage. If of recent origin, the cartilage included within the circumference of this fissure may differ little from that of the rest of the articular surface, but it is often somewhat opaque, having lost the normal transparent bluish appearance, while it may be roughened, sodden, and even stained by the blood-pigment in the synovial fluid. At one aspect of the circumference, often towards the mesial side, the cartilage is unbroken, here forming a hinge by which the osteo-cartilaginous fragment is held more or less in its original position. A

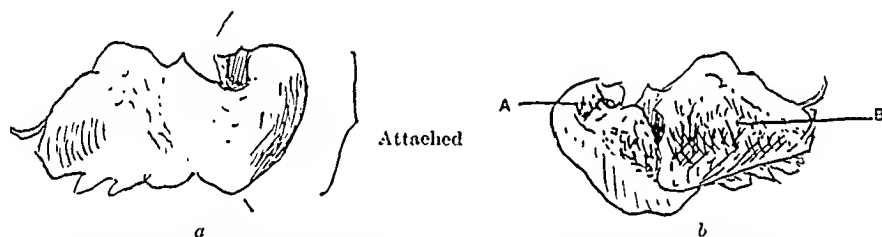


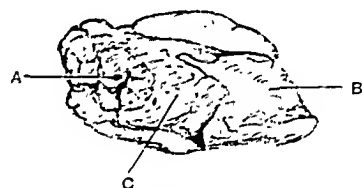
FIG. 54.—Fragment of cartilage, calcified cartilage (B), and bone (A) removed from knee seen in *Figs. 52, 53*, showing shaggy irregular margins. In spite of a long history the detachment appears to have been recent. *a*, Superficial surface. *b*, Deep surface. (*Actual size*.)

flat dissector, introduced through the fissure, can be moved freely beneath the loosened fragment. Division of the cartilaginous hinge sets the fragment free and reveals the depression in which it lay. If of recent origin, the margins of this hole are abrupt, clean-cut, and slightly shelving, with perhaps some undermining of the cartilage here and there. The bone in the floor presents a slightly concave surface covered by a thin layer of sodden fibrous tissue. If the lesion is of long standing, some proliferation of the surrounding cartilage will have occurred, the margins of the hole then being rounded off and the floor invaded to some extent by fibrocartilage. The greatest amount of repair will be seen when the fragment has been completely detached for some considerable time before operation. The appearance of the fragment removed also varies with the duration of the lesion. If operation has been resorted to early, but little change will be seen, the fragment consisting of a convex layer of more or less normal cartilage, with slightly frayed abrupt margins, and with a piece of dead bone, of smaller area, adherent to its deep surface (*Fig. 54*). The cartilage may be fractured across the middle of the fragment. If sufficient time has elapsed, the margins of the fragment will be rounded off, and the bone on its deep aspect may be partially covered as

a result of proliferation of the overlying cartilage (*Fig. 55*). These changes are particularly well seen when the detached fragment includes a portion of the intercondylar notch and is anchored by a pedicle of synovial membrane in which may be present a few fibres of the posterior crucial ligament. In this case the bone in the fragment is living and may show evidence, under the microscope, of new bone formation.



FIG. 55.—Fragment of cartilage and bone removed, four months after injury to foot, from knee shown in *Fig. 45*. Fragment partially loose and stained with blood-pigment. The margins have become somewhat rounded off. A, Bone; B, Cartilage; C, Fibrous tissue. (*Actual size.*)



Such are the common findings. There are others, however, which are less frequently met with. In spite of the typical radiogram the cartilage may be found unbroken, as in Mr. Bristow's case. The site of the lesion may still be obvious, however, the cartilage being discoloured, roughened, sodden, and outlined by a line or slight groove. Though the surface is unbroken, this portion of cartilage may be slightly but quite definitely mobile over the underlying bone. On incising around such an area, the fragment, including some bone, may be found completely undermined and loose, as reported by Richards, Frieberg, and Ludloff.¹¹ On the other hand, in spite of a typical radiogram, incision of the unbroken cartilage may reveal no loose fragment. In our series a femoral case of this type required the slight use of a gouge to complete the separation of the bony fragment (*see Figs. 45, 55*), while in a lesion of the patella, on the other hand, there was no suggestion of the bone being loose (*Fig. 56*). Where the fragment includes the condylar margin and has become completely detached, one occasionally sees the resulting crater occupied by a polypoid growth from the soft tissues in the intercondylar notch. As is sometimes seen after traumatic lesions of the articular margin elsewhere in the joint, this polyp may be fibrocartilaginous in



FIG. 56.—Lateral skiagram of knee showing fragment, apparently loose, lying in crater in patella. At operation fragment found quite firm. Girl, aged 14.

consistence. It is not very uncommon, however, to see lesions of the articular cartilage only in the typical situation on the inner condyle, when, for one reason or another, a knee-joint with a normal X-ray is explored. The cartilage may be simply roughened or pitted; its surface may be soft and sodden; over a limited area it may be movable on the underlying bone; a thin flake

not involving the whole thickness of the cartilage may be raised and hanging by a hinge; or, lastly, a fragment of cartilage may be completely detached from the typical site, leaving a sharp-edged crater with a *convex* floor formed by the undamaged bone of the condyle.



FIG. 57.—Free fragment of normal cartilage only, with abrupt recently fractured margins, removed from intercondylar notch. Woman, aged 24. Five years' history of trouble in knee. Recent injury. Knee locked. Released under gas. *a*, Superficial surface. *b*, Deep surface. (Actual size.)

Such a condition as the last was revealed in a young woman who was carried off the tennis court with a locked knee. Two weeks later a free flake of articular cartilage, apparently recently detached and showing abrupt

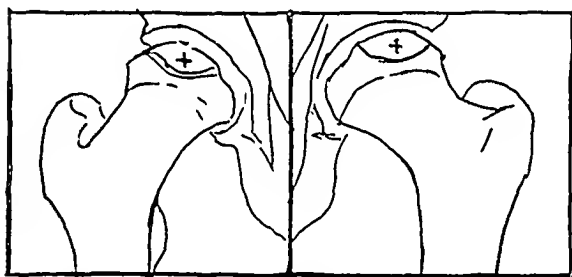


FIG. 58.—Bilateral osteo-chondritis dissecans of head of femur. (After Lange.)

margins, was removed from the intercondylar notch (Fig. 57). The X-ray was negative, but at the operation the crater from which this flake had been detached was seen on the inner condyle at the usual spot. The joint contained dark blood. There was a history of the knee having been twisted while skiing five years before, with some trouble on four occasions since. In spite of this history, the evidence pointed strongly to the lesion being the direct result of the recent trauma.

In the *elbow*, after trauma, we are all familiar with obvious and varying X-ray changes in the capitulum, short of complete displacement of this bony prominence. A study of the elbow cases is complicated by the occurrence of what is believed to be a non-dissecting type of osteo-chondritis of the capitulum, and I do not propose in this paper to embark on a consideration of the difficult problem presented by this joint.

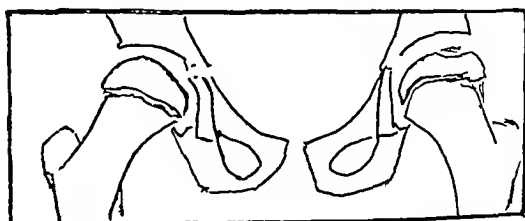


FIG. 59.—Unilateral osteo-chondritis dissecans of head of femur. (After Gold.)

Osteo-chondritis dissecans of the *hip-joint*, of which I have no personal experience, seems to be on the same plane as the elbow. The lesions said to be typical in this joint are well seen in the radiograms of a bilateral and a unilateral case published by Lange¹² and Gold¹³ respectively (Figs. 58, 59).

They should be compared, however, with the very suggestive appearances seen in the head of the right femur in the radiogram of a boy of 6 (*Fig. 60*). This small lesion did not progress, but cleared up in the course of a few

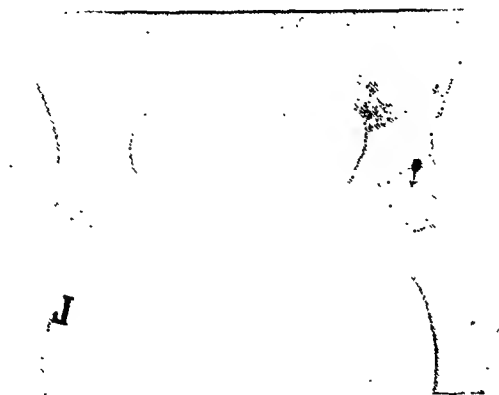


FIG. 60.—Boy, aged 6. Right hip shows small lesion at highest point of head suggestive of lesion said to be typical of osteo-chondritis dissecans of hip-joint. Left hip shows typical pseudo-coxalgia.

months. The other hip progressed through all the changes typical of pseudo-coxalgia. This second case seems to show a similar contrast in the X-ray shadows thrown by the two hips (*Fig. 61*).



FIG. 61.—Boy, aged 7. Right hip shows lesion in head of femur similar to osteo-chondritis dissecans. Left hip shows early change of pseudo-coxalgia.

A lesion of the type under discussion affecting the *ankle-joint* I have met with only once, and then in a young woman, kindly referred to me by Mr. Tyrrell Gray (*Figs. 62—64*). There was a definite history of trauma.

Exploration revealed a typical clean-cut osteo-cartilaginous fragment partially detached from the saddle surface of the astragalus, and only held in place by a cartilaginous hinge behind.



FIGS. 62, 63.—Osteo-chondritis dissecans of astragalus. Young woman, with history of trauma. Note fragment partially detached from superior articular surface of astragalus.



FIG. 64.—Fragment removed from case shown in Figs. 62, 63. *a*, Superficial surface. *b*, Deep surface. (Actual size.)

Though several writers mention the *shoulder*, I have not found any detailed reference to a typical lesion of this joint.

ETIOLOGY.

Though Broca,¹⁴ Teale,¹⁵ and Paget¹⁶ had previously and independently suggested that the separation was effected by a pathological process—a 'quiet necrosis'—following trauma, König's¹⁷ name is usually associated with this theory. He gave to the condition the name by which it is generally known to-day, but admitted that all signs of inflammation had disappeared. The majority of surgeons accept trauma as playing some part at any rate in the etiology, if only as the cause of the final complete displacement of the fragment. It will be as well, therefore, to consider first how trauma can possibly occur at the typical spot on the inner condyle. Direct injury from without can be ruled out, since at the site of election for these lesions the articular surface is well protected in all positions of the joint. The blow or blows must be struck by one of the other two bones forming the joint. Two theories receive support: first, that the patella is responsible, and second, the tibial spine.

Now the patella normally comes in contact with this particular portion of the inner condyle only when the joint is fully flexed, and this is surely a very unusual position for the knee when the latter is traumatized. It seems quite out of the question that the patella could damage the inner condyle at the site of election without the assistance of external violence, and then only if the joint were flexed beyond a right angle. Then again, if the patella is commonly responsible, one would have expected the flexed position of the joint to be mentioned in the histories of some of the cases at any rate. Brackett,¹⁸ it is true, cites a case of the typical lesion resulting from a severe blow on a fully flexed knee, and calls attention to the fact that many of his patients followed occupations involving an excessive amount of kneeling, but this does not seem to be the experience of most writers. Hellström, however, also blames the patella, believing the lesion to be an incomplete fracture resulting from repeated minor traumata, the damage occurring when the joint is flexed.

Professor Blair, to whom I am much indebted for helpful criticisms, made the interesting suggestion that, since kneeling puts tension on the posterior cruciate, excessive kneeling might induce an abnormal development of the inner tubercle of the tibial spine—to which this ligament may be partially attached—and thus indirectly favour the occurrence of the lesion. As regards the patella itself, in several of our cases (7 of 20), the deep surface has shown signs of damage. The cartilage has been split, pitted, shredded, and sodden, but not as a rule at its inner margin. These changes are not, however, invariably present, and are seen quite commonly without a condylar lesion; I have always regarded them as either secondary to the irregularity of the surface of the condyle or as independent traumatic lesions. Though admitting, of course, that a subluxating patella can damage the outer condyle, I think it extremely improbable that this bone is responsible for the lesions on the inner, except in very rare cases such as Brackett's.

It seems to me easier to accept the theory that the tibial spine is responsible. The inner appears to be the larger of the two tubercles in most knees, if not in all, but certainly in those affected by osteo-chondritis dissecans. Several writers have called attention to what they regard as the excessive size of the inner tubercle in their cases. There are two ways in which this tubercle may be forced against the inner condyle—namely, by rotation of the tibia on the femur (or vice versa) or by an external shearing force driving the tibia inwards or the femur outwards. It is difficult to obtain a clear account of the accident in cases with a definite history of trauma, but it seems probable that in most cases at any rate, the first suggestion, that of rotation, is correct. Roesner¹⁹ favours the rotation theory, but speaks of the involuntary rotation, i.e., *external* rotation, which takes place just before extension is completed. In the cadaver he was able to produce lesions of the articular cartilage at the typical site by forced external rotation of the tibia.

In the hope of throwing light on this question, a number of dissected specimens were examined with Professor Blair. We found that as a rule impingement of the tibial spine against the inner condyle was produced by *internal* rotation of the tibia on the femur. Only in one of the specimens

examined was it evident that external rather than internal rotation of the tibia brought the spine to bear against the condyle.

Study of the lateral radiograms of affected knees suggests that the amount of flexion present at the moment of rotation must be very small. Frieburg says it is easy to make the spine impinge on the posterior crucial ligament, even when the inner tubercle is not unduly long. Barth,²⁰ who favours the pure traumatic theory, suggests that the pull of this ligament is responsible, but this could hardly explain the numerous cases in which the margin of the intercondylar notch is not involved in the lesion.

Now let us pass to a consideration of the various theories that have been suggested to explain the occurrence of the lesions.

Trauma Pure and Simple.—This means the lesion is a fracture. This theory receives the support of several writers, though they do not all agree as to the mechanism by which the injury occurs (Brackett, Richards, Barth, Roesner, and Kappis²¹). Since the evidence, it seems to me, is overwhelming that this theory is correct, it will be convenient to deal with the other theories first.

Trauma Followed by some Pathological Process.—We have already noted that the early writers thought the separation was the result of an inflammatory process set up by trauma—a 'quiet necrosis'. Unfortunately there is no evidence whatever of inflammation, present or past. The vast majority of investigators agree on this point. Mouchet and Bruas²² speak of a 'diaclastic action' following trauma, by which they probably mean very much the same thing. The only alternative is that the blood-supply to a portion of bone is destroyed, directly or indirectly, by a trauma, and the resulting dead fragment is separated in the usual way, possibly with the help of additional traumata, but at any rate with a minimum of inflammatory reaction. If the vessels are severed by a crack in the bone, i.e., the fragment is broken off, as I believe it is, there is no need to invoke any obscure process to explain the death of the bone and its separation. Is it possible that the bone at the site of the lesion, though not actually fractured, is so bruised or otherwise damaged that necrosis occurs, without the addition of infection? This seems most improbable.

Somner²³ suggests that trauma, perhaps slight and oft-repeated, causes paralysis of the local vessels. Roesner found 'crushed trabeculae', but this would agree with the pure traumatic theory better than with any other. Ludloff and Frieburg attribute the lesion to 'infarction' resulting from damage to the vessels entering the bone from the intercondylar notch. The former calls attention to a vessel, the *arteria genu media*, the branches of which ramify over the posterior crucial and the inner condyle, and which, he says, is often a terminal artery. If injury to this vessel were really the cause, one would expect to find the margin of the intercondylar notch included in the fragment with some regularity, whereas we know that this only occurs occasionally. Moreover, Nussbaum²⁴ affirms that this vessel is not an end artery. An even more convincing argument against this theory, as Phemister points out, lies in the fact that when the fragment includes the margin of the condyle and is pedunculated by the crucial ligament and synovial membrane, the bone in the fragment is not dead but living, and may show evidence of new

bone formation. How can this fact be reconciled with any theory which presumes necrosis of the fragment before its separation? If the fragment is a sequestrum, cast off in the usual manner, where is the granulation tissue essential for this process? Though some describe the floor of the crater as being covered with granulations, I venture to assert that it is quite exceptional to find the cavity lined by tissue which could in any way justify this description. Even when the history and the condition of the margins of the fragment and crater indicate a recent separation, the latter is covered by nothing but the thinnest layer of fibrous tissue. Occasionally this tissue may be rather thicker and more vascular in places, but very rarely, in my experience, does it deserve the name of granulation tissue. Axhausen,²⁵ however, when operating soon after an injury found indications of a process long antedating the final trauma. He found resorption, very active connective tissue, and giant cells, eroding necrotic bone. König, as already stated, found no sign of inflammation. Hellström and Phemister both say the same. Paire and de Bourguet²⁶ say that inflammatory exudate and cells are never found. Somner found no sign of inflammation in any of his twelve cases.

Wolbach and Allison's²⁷ post-mortem specimen, which they reported so carefully, was unfortunately complicated by a cyst in the condyle adjacent to the typical lesion, and was, therefore, quite exceptional. Any changes found in the thin layer of bone between the crater and cyst in their case are, I contend, useless so far as our discussion is concerned. They found the floor of the crater, however, covered with the usual layer of fibrous tissue with some cartilage cells. They remark on the absence of repair such as is seen in experimental incision of a bone or in a fracture; but are not the above signs of repair, and all that were to be expected on a surface bathed by synovial fluid and probably traumatized frequently by the fragment, which in their case was pedunculated? Opportunities such as this for complete examination of a crater and the bone beneath it must be extremely rare. On the other hand, for investigation of the loosened fragment there is an abundance of material. Microscopic examination of these fragments or loose bodies, it seems to me, shows only those changes which are known to occur in any osteo-cartilaginous fragment separated from an articular surface by direct trauma. If the blood-supply has been entirely severed, the fragment being loose or only held in place by a portion of unbroken cartilage, the bone is dead, though otherwise normal, while the cartilage is living and may show signs of proliferation. If a vascular pedicle was present, the bone as well as the cartilage may be alive, and if sufficient time has elapsed, there may be evidence of newly-formed calcified tissue and bone. If a subsequent trauma has completed the separation, the bone, though modified as above, will be dead. I have failed to find any report of findings which are not consistent with the separation having been effected by trauma, and trauma alone.

The Embolic Theory.—Axhausen,²⁸ who at first was a strong advocate of the 'damage-to-vessels' theory, is responsible for another suggestion which has received a fair measure of support, and which attributes the lesions to embolic infarction. He suggests attenuated tubercle bacilli as producing the occlusion of the vessels, but why such emboli, of tubercle or any other bacilli, should be found in healthy young people it is difficult to conceive. No proof

is forthcoming that such embolism occurs in these cases. If such is possible, why should the inner condyle be selected, occasionally even in both knees, and the usual sites in a bone for acute or chronic infection escape? Paitre and de Bourguet, supporting this theory, invoke a 'special fragility' of the epiphysis. Rieger²⁹ suggested fat embolism, and even stated he had found actual evidence of this, but no one has confirmed his improbable findings. The fact that when pedunculated the fragment consists of *living* bone and cartilage affords as strong an argument against this theory as it does against all the others with the exception of pure trauma. In this connection it is of interest to recall the experiments of Phemister, who killed portions of bone by insertion of radium into joints, and found that the necrotic fragments were not exfoliated, and moreover they gave rise to no inflammatory reactions, such as might have been expected, in the adjacent living bone.

Other Theories.—Bernstein³⁰ cites a bilateral knee case, a boy, with two sisters suffering from a similar lesion in one knee, and suggests an hereditary constitutional influence. Wagoner and Colm,³¹ of whose five cases three were members of the same family, also stress the hereditary factor. Weil³², in view of the bilateral cases, speaks of it as a generalized disease.

Is it not at least as easy to assume, in these knees, an anatomical peculiarity present in all of them and favouring trauma at the typical site for these lesions? Archer and Peterson³³ suggest it is an adolescent affection similar to Perthes' disease, while Axhausen also draws a comparison between this condition and the familiar forms of osteo-chondritis. I have already admitted the difficulty of differentiating lesions of one type of osteo-chondritis from

those of another in the elbow- and hip-joints. But the typical lesions in these joints, and also in the knee, are occasionally seen in cases long past the age for other forms of osteo-chondritis. My oldest knee case was 45, while two others, in one of which the fragment contained cartilage only, were over 40.



FIG. 65.—Lateral skiagram showing large fragment broken from posterior part of outer condyle, result of twist of joint. Man, aged 30.

I feel forced towards the view that all these latter theories are no more than theories with little or nothing to support them, and that in considering them we are going out of our way to find an explanation when we have trauma as a possible, and, in my opinion, probable, explanation of all the various conditions described above. Somner found a definite history of trauma in 7 of his 12 cases. In our own series, three-quarters gave a history of trauma—a blow

or kick on the knee in 5, and a twist, football accident, etc., in 10; in 2 the history was indefinite; while in 4 only was there definitely no trauma. As already stated, the fragment may consist of nothing but *avascular* cartilage. By what possible process, other than direct trauma, can such a lesion occur in a joint which otherwise shows no change? That a considerable fragment can be broken off a condyle by a man's unaided efforts is proved by this

skiagram (*Fig. 65*). The patient, a man of 30, swung round somewhat abruptly to the right, to reach a book from a shelf behind him, his right foot being firmly planted on the floor. There was no question of external violence. Four weeks later a large fragment, quite free and obviously the result of a recent fracture, was removed from the back of the joint. Presumably this piece of the outer condyle was knocked off by the external tubercle of the tibial spine. The popliteus tendon was not attached to the fragment, which consisted of nothing but articular cartilage and bone.

As to those cases in which a typical lesion is seen in the radiogram and yet the cartilage is found intact, I can conceive it quite possible, as Hellström suggests, that the resilient cartilage, though bruised and damaged by a blow, might remain unbroken, and yet a fracture occur of the resistant and brittle bone beneath. Kappis speaks of a 'subchondral hæmatoma' being produced—surely quite a possible result of trauma, with or without a fracture of the bone. Panner³⁴ draws a rather attractive comparison with those cases of fracture of the carpal scaphoid in which the crack, seen only with the greatest difficulty soon after the injury, becomes obvious within a few weeks. Such a crack might easily be overlooked in the femoral condyle in a radiogram taken shortly after an injury. If a subchondral fracture occurs, it is quite possible for union to take place provided the movement of the fragment in its bed is minimal. Moreau³⁵ and Kappis both state that complete cure can occur with disappearance of the characteristic X-ray picture. If, however, the patient continues to indulge in violent exercise, the movement of the fragment may be more than minimal, and the fracture may remain ununited. Even when operation is delayed, therefore, the bony fragment may be found to be loose in spite of the cartilage being intact.

It remains for me to suggest an explanation of the lesion being found in the absence of a history of trauma. Supposing the initial damage left the cartilage unbroken, there would be no rough surface and no escape of blood to irritate the joint, and possibly no synovial effusion of any kind. It seems just possible that, in the heat of a game, the pain caused by the injury might attract but little attention, while later there would be no discomfort. Repeated subsequent traumata, perhaps not very severe, might break the cartilage and even gradually complete the separation of the fragment. Phemister makes the rather surprising assertion that the subchondral bone is peculiarly insensitive. He bases this statement on his experience when operating under local anæsthesia, saying that he had "chiselled off marginal osteophytes and small chips of cartilage and bone from the articular surface, apparently without producing any pain." It is evident, however, that he was dealing with grossly abnormal articular surfaces. Professor Blair was again consulted, but little work seems to have been done on the nerve-supply of bone. Further research is necessary before we can answer this question of sensitivity of subchondral bone.

To sum up, I would suggest that the typical lesion is a fracture and nothing else, for the following reasons:—

1. It most frequently occurs in adolescents and young adults indulging in vigorous pastimes.

2. Typical lesions are seen in radiograms and revealed by operation after definite trauma, which in some cases is quite recent.

3. A lesion at the typical site may involve the cartilage only, the detached fragment consisting of normal articular cartilage. In such cases there is a definite history of trauma.

4. There is an entire absence of any inflammatory changes, macroscopic and microscopic, in or about the lesions.

5. The naked-eye appearances, when operation is performed early, suggest nothing but a simple recent fracture. When sufficient time has elapsed for changes to occur, they are only those which we should expect as the result of an effort on the part of the tissues to repair the damage. Precisely similar changes are occasionally found on the more exposed parts of the femoral articular surface, when the traumatic origin of the lesions is never disputed.

6. When the detached fragment is suspended by a vascular pedicle the bone in it is not dead, and is not a sequestrum, so why should it have been exfoliated?

7. To explain the occurrence of the lesion in both knees, or in the knees of more than one member of a family, it is easier to accept the presence of anatomical peculiarities which favour exceptional local trauma than the suggestion of embolism, damage to blood-supply, or indeed any other theory.

TREATMENT.

Probably all will agree that in the absence of symptoms the finding of a typical lesion in a radiogram is not sufficient excuse for opening the joint. However, it is extremely unlikely that this discovery will be made except in the course of routine X-ray examination in a bilateral case, admittedly a very rare experience. I have found the lesion unexpectedly on routine X-ray examination in cases with a damaged semilunar cartilage. As already stated, disappearance of the lesion is possible. Hellström, however, advises operation in spite of the absence of symptoms, since otherwise, he says, osteo-arthritis will develop. This late complication cannot, I fear, be avoided, even by early operation.

In the presence of symptoms the joint should always be explored. If, on opening the joint, the articular surface is found to be unbroken, but the site of the lesion is clearly indicated by alteration in the colour or texture of the overlying cartilage, or the extent of the lesion is indicated by a groove, an attempt should be made to determine whether this circumscribed area of cartilage is movable or not. If it is, there seems no doubt about the wisdom of excising it, together with any loose bone beneath it. If the cartilage does not move over the bone, the problem is more difficult. Some surgeons I know would prefer to leave well alone. Personally I feel that the exact condition of the cartilage within the circumference of the lesion should be the determining factor. If definitely soft, sodden, and rough, although unbroken, I think it is better excised. Any bone found loose should be removed and the edges of the hole carefully bevelled. If, on the other hand, the cartilage is almost normal in appearance and the lesion

only just discernible, and there is nothing to suggest that a fragment of bone beneath is loose, it may safely be left alone. If in doubt, and particularly if the mobility or otherwise of the fragment beneath is uncertain, in my opinion it is wiser to excise the lesion. I should prefer to err on the side of radical treatment.

If the lesion presents the more usual appearance with the cartilage fractured, but with an unbroken portion holding the fragment more or less in position, the separation should be completed and the fragment removed. The cartilaginous margins of the crater should be carefully bevelled where necessary, and any undermined portions removed. I see nothing to be gained by curetting or otherwise treating the floor. There is no object in making the crater deeper. Any filling in that occurs will be done for the most part by the margins of the cartilage, and not by the bone, even though the latter be freshened by gouge or spoon. If the fragment is free in the joint and the X-ray shows the site from which it came, the incision should be planned to allow for inspection of the crater as well as removal of the loose body. In all cases the condition of the semilunar cartilages should be determined. In three of our cases a lesion of a semilunar cartilage (internal two, external one) was found in conjunction with the typical damage to the internal condyle.

The immediate *prognosis*, and for some years to follow, is undoubtedly good. The remote results one would hardly expect to be so favourable, and there is reason for believing that osteo-arthritic changes are certain to occur sooner or later.

SUMMARY.

In the above remarks I have briefly reviewed the known facts of the condition with special reference to the knee-joint, and have endeavoured to show that these point strongly to trauma, pure and simple, as the cause of the characteristic lesions. The mechanism of this trauma in the case of the knee-joint has been discussed, and it has been suggested that violent rotation inwards of the tibia, driving the tibial spine against the inner condyle, is responsible for the injury in most cases. I have ventured to give my own personal views as to the treatment suitable to the varying conditions which may be found on exploring the joint.

REFERENCES.

- ¹ PHEMISTER, *Jour. Bone and Joint Surg.*, 1924, April, 278.
- ² RICHARDS, *Amer. Jour. of Roentgenol.*, 1928, xix, 278.
- ³ BALENSWEG, *Jour. Bone and Joint Surg.*, 1925, vii, 465.
- ⁴ DELCHEF, *Arch. franco-belges de Chir.*, 1927, April, 331.
- ⁵ HEINE, *Deut. Zeits. f. Chir.*, 1927, cccvi, 119.
- ⁶ NIESSEN, *Beitr. z. klin. Chir.*, 1928, cxliii, 159.
- ⁷ HELLSTRÖM, *Acta Chir. Scand.*, 1922, lv, 190.
- ⁸ AXHAUSEN, *Arch. f. klin. Chir.*, 1914, civ, 581.
- ⁹ JANSSON, *Acta Radiol.*, 1930, xi, 33.
- ¹⁰ FRIEBURG, *Jour. Bone and Joint Surg.*, 1923, Jan., 3.
- ¹¹ LUDLOFF, *Arch. f. klin. Chir.*, 1908, lxxxvii, 552.
- ¹² LANGE, *Zeits. f. orthop. Chir.*, 1929, March, 268.

- ¹³ GOLD, *Zeits. f. Chir.*, 1930, cxxv, 296.
- ¹⁴ BROCA, quoted by Phenister.
- ¹⁵ TEALE, *Med.-Chir. Trans.*, 1856, xxxix, 31.
- ¹⁶ PAGET, *St. Bart.'s Hosp. Rep.*, 1870, vi, 1.
- ¹⁷ KONIG, *Deut. Zeits. f. Chir.*, 1888, xxvii, 90.
- ¹⁸ BRACKETT, *Amer. Jour. Orthop. Surg.*, 1917, Feb., 79.
- ¹⁹ ROESNER, *Beitr. z. klin. Chir.*, 1922, cxxvii, 537.
- ²⁰ BARTH, *Arch. f. klin. Chir.*, 1898, lvi, 507.
- ²¹ KAPPIS, *Deut. Zeits. f. Chir.*, 1920, clvii, 214.
- ²² MOUCHET and BRUAS, *Lyon Chir.*, 1925, xxii, 510.
- ²³ SOMNER, *Beitr. f. klin. Chir.*, 1923, cxxix, 1.
- ²⁴ NUSSBAUM, *Ibid.*, 244.
- ²⁵ AXHAUSEN, *Deut. med. Woch.*, 1920, July 22, 825.
- ²⁶ PAITRE and DE BOURGUET, *Rev. de Chir.*, 1927, lxx, 626.
- ²⁷ WOLBACH and ALLISON, *Arch. of Surg.*, 1928, xvi, 1176.
- ²⁸ AXHAUSEN, *Münch. med. Woch.*, 1922, lxix, 881; *Beitr. z. klin. Chir.*, 1922, cxxvi, 451.
- ²⁹ RIEGER, *Münch. med. Woch.*, 1919, June 15.
- ³⁰ BERNSTEIN, *Jour. Bone and Joint Surg.*, 1925, vii, 319.
- ³¹ WAGONER and COHN, *Arch. of Surg.*, 1931, July, 1.
- ³² WEIL, *Beitr. z. klin. Chir.*, 1913, lxxvii, 403.
- ³³ ARCHER and PETERSON, *South. Med. Jour.*, 1930, xxiii, 611.
- ³⁴ PANNER, *Acta Radiol.*, 1924, iii, 129.
- ³⁵ MOREAU, *Arch. franco-belges de Chir.*, 1923, Feb., 131.

THE ANATOMY OF THE PERIPHERAL SYMPATHETIC NERVOUS SYSTEM.

By H. H. WOOLLARD AND R. E. NORRISH,

ANATOMY DEPARTMENT, IN ASSOCIATION WITH THE SURGICAL UNIT, ST. BARTHOLOMEW'S MEDICAL COLLEGE.

ILLUSTRATIONS of the peripheral portion of the sympathetic nervous system have been designed either to show the physiological implications of its structure or to convey what appears to be the amazing complexity of its arrangements. Now that surgical resections of this system have become frequent, numerous publications bearing on its anatomy have appeared, and these seem to introduce a new kind of complexity. The complexity of the earlier representations arose from the picturing of rich arborizations amongst all the branches of the sympathetic nerves so that there resulted a bewildering interplexiform arrangement making it wellnigh impossible to determine how any particular organ might be deprived of its sympathetic innervation by surgical methods. The new complexity arises from the implication that there is no constant and formal pattern in the source of supply to the viscera, in that these may receive their innervation from adjacent plexuses or from branches proceeding from the ganglia, and further that a third source exists in the fibres which accompany the blood-vessels.

The object of the present paper is to review these suggestions and possibilities, and to determine with as much precision as possible the anatomical routes the nerves pursue, and how these can be approached and resected by the surgeon.

The well-known picture of Hirschfeld emphasizes the apparent complexity of the sympathetic nervous system. This picture has been used over and over again in various text-books and often modified to express some particular point of view. In the *Atlas* of Spalteholz and in Quain's *Anatomy* the representations of the sympathetic nervous system appear to be less complicated, and indeed we think our own investigations are more in accordance with the figures which appear in these books.

COMPARATIVE ANATOMY.

In order to grasp the morphological principles which underlie the peripheral distribution of the sympathetic system, it is necessary to refer briefly to its comparative anatomy and embryology.

The comparative anatomy of the system is not very well known, but what little information we have at our disposal goes to show that this system is a fairly late acquisition in the history of the animal kingdom. In *Petromyzon*, the most primitive of the chordates, a vagal system can be distinguished. Later comes an adumbration of the sacral outflow of

parasympathetic fibres. Then later in the history of the vertebrates the thoraco-lumbar portion of the sympathetic system proper makes its appearance. Therefore it is to be inferred that the autonomic system as a whole appears comparatively late, and of its three portions the sympathetic system (keeping this term for the thoraco-lumbar outflow) is the last of all to appear. It is interesting to observe that the same order is repeated in development, and in mammals the thoraco-lumbar portion of the autonomic system is the last to make its appearance.

As is well known, some of the cells belonging to the sympathetic nervous system become differentiated into adrenalin-producing cells. The totality of these cells which can liberate the sympathomimetic adrenalin is known as the 'chromaffin system'. The comparative anatomy of the sympathetic nervous system reveals that the chromaffin system is much more extensive in the lower vertebrates than in ourselves, and that it undergoes a progressive reduction as one passes up the animal scale. With this reduction in the extent of the chromaffin system there is a progressive increase in the peripheral plexuses of the sympathetic system. It would seem that in man the chromaffin system is still undergoing reduction, for those outlying chromaffin masses that lie beside the aorta and adjacent to the suprarenal, the para-aortic bodies of Zuckerkandl, disappear in the first few years after birth. The conversion of sympathetic medulloblasts into chromaffin-secreting cells has been a long-established embryological fact, and when Elliott established also that all fibres proceeding to the medulla of the suprarenal were preganglionic, it became clear that the rôle of adrenalin was the replacement of a postganglionic fibre by the distribution through the blood of a chemical substance. Obviously this method produces a diffuse response and is much slower than the more localized and swifter-moving nerve impulse. Thus it is clear, from the inverse relationship between these two modes of distributing sympathetic effects, that in the course of evolution the less exact has given way to the more precise and more highly differentiated method.

There are morphological reasons for suggesting that the visceral centres were first laid down in the hypothalamus, and exerted their effects on the periphery by means of the release of a chemical substance, and that out of this system has grown the peripheral sympathetic nervous system. Owing to its late arrival, parts of the neural axis had been already allotted to the extremities, and therefore it has become restricted in its outflow from the central axis to a range between the first thoracic and the third lumbar segment, and moreover its connector neuron became displaced to the periphery, for the only available source of neural proliferation was the neural crest. However, be this as it may, it is from the neural crest that cells of the sympathetic ganglia and the chromaffin cells take origin, thus arising from the same source as the dorsal ganglia of the cerebrospinal nerves.

The adrenalin produced in the chromaffin cells not only acts as a substitute for the fibre starting from the peripheral ganglion—that is, the postganglionic fibre—but also acts as an intermediary between the ending of the postganglionic fibre and the contractile or secreting element which it innervates. It seems possible that an adrenalin-like substance (called 'sympathin' by Cannon) does participate in every reaction produced by the sympathetic

fibres and is indeed the agent which produces the reaction. The fact that all sympathetic endings are pericellular and not intracellular is consistent with this idea. Therefore it is to be supposed that although the adrenalin as a replacer of sympathetic fibres is less in evidence in man than in lower vertebrates, the same does not obtain in the case of adrenalin or adrenalin-like substance acting as an intermediary between the nerve-ending and the reacting tissue.

The neuroblasts which are proliferated from the neural crest become aggregated in a segmental manner, so that in the postganglionic system of the sympathetic the metameric arrangement is preserved. A segmental plan does not occur in the preganglionic system, for this part, as is well known, is restricted roughly to the thoracic region of the cord. Its connections with the peripheral ganglia therefore show an overlapping. The extent and nature of this overlap was studied minutely by Langley, and from his work it can be concluded that each preganglionic fibre is likely to be connected with somewhere between three and six peripheral ganglia. This suggests that activity starting centrally is likely to have widespread effects at the periphery.

The peripheral distribution of the postganglionic fibres diverges into two great streams, one directed cranially and one directed caudally. The separation between the two systems is about the tenth thoracic segment. Those that pass cranially innervate the head and face, the eye, the heart, the lungs, and the upper extremity, in that order cranio-caudally. This is also the cranio-caudal order in which these structures arise in the course of development. The same is to be observed in the caudal region, for the succession in the sympathetic outflow proceeds to the abdomen, the pelvis, the lower limbs, and the perineal region.

To some extent morphological principles are exhibited in the peripheral fibres when their distribution is studied in relation to the emergence of the organs of the body in a transverse plane. As is well known, the trunk of the embryo in the earlier stages of its development shows three main regions in which are differentiated certain organ-systems of the body. These are distinguished most laterally as the somatopleure, from which the parietes of the abdominal wall are developed; in the median plane is found the splanchnopleure, which surrounds the entoderm of the gut; and between these dorsally a ridge of mesenchyme—the intermediate cell mass or the Wolffian ridge—in which the suprarenal, the kidney, and the gonad are developed.

Regarding the distribution of the sympathetic fibres to these areas, those for the parietes reach their peripheral destination by travelling in the spinal nerves. Those fibres which go to the structures derived from the Wolffian ridge come from a laterally placed ganglion—the renal ganglion, which is also the source of the fibres to the testis or ovary. The median entodermal structures have their ganglia piled along the front of the aorta, and the fibres from these accompany the blood-vessels to their end structures.

These three systems, which can be broadly distinguished morphologically, are also different physiologically. The parietal and limb fibres are vasomotor, sudomotor, and pilomotor; those to the intermediate structures, like the kidney and the testis, have no special function, for as yet nothing beyond

vasomotor effects has been discovered ; those going to the mid-line structures are for the most part of an inhibitory nature, depressing secretion, inhibiting peristalsis, and dilating bronchioles ; they are, of course, tonic to the sphincters.

In the abdomen this distinction between the three distributive arrangements can be apprehended most easily. The segmental ganglia are the source of the limb fibres, the intermediate organs have their own special group, while the great pre-aortic structures function for the alimentary system. The activity of the parietal group produces tonic constrictor effects, while the medial system is both vasoconstrictor and vasodilator to the blood-vessels, while being inhibitor to the circular and longitudinal muscle coats of the bowel.

From this brief review of the sympathetic system it can be inferred that it follows definite morphological principles, and that in its peripheral distribution we may hope to be able to determine well-marked and definite courses of fibres so arranged that it can be said that their removal would result in the denervation of precise regions of the body. This view is to some extent in conflict with the work of other anatomists, whose investigations have led them to emphasize the syncretic aspects of the sympathetic systems. Nor does it readily harmonize with the work of Kondratjew, who has emphasized the existence of interorganal plexuses between adjacent viscera within the abdomen.

We would now proceed to discuss the anatomy of the sympathetic system in some of the regions of more immediate interest to the surgeon.

THE SYMPATHETIC INNERVATION OF THE EYE.

The nodal point in the origin and distribution of the peripheral fibres to those parts of the body above the diaphragm is the ganglion stellatum. By this we mean the inferior cervical ganglion and the first thoracic. These are so often fused and the distinction between them is often so slight that the term 'ganglion stellatum' can be used quite justifiably. This ganglion, along with the outlying middle and superior ganglia, really represents fused segmental and prevertebral ganglia—that is, it has the combined significance of, say, the lumbar and the celiac ganglia in the abdomen. From it proceed segmental rami as well as visceral branches such as the cardiac accelerators. It was for these reasons that Gaskell was tempted to apply the term 'superior splanchnic' to the cervical sympathetic.

From the stellate ganglion the peripheral fibres directly or indirectly proceed farther. Of these fibres some will proceed as preganglionic fibres to the middle and superior cervical ganglia ; others, the majority, will emerge from it as postganglionic fibres. Those which proceed to the eye, for instance, pass into the cervical sympathetic, traversing the stellate ganglion and then passing in front of the subclavian artery. When they reach the superior cervical ganglion they enter into synaptic arrangement with the cells of this ganglion. Thereafter they reach the cavernous plexus by travelling with the internal carotid, and from there reach the extrinsic unstriated muscle of the orbit as well as the dilator fibres of the pupil. On these they exert a continuous

tonic effect, for as soon as they are interrupted the well-known phenomena of Horner's syndrome make their appearance.

These ocular sympathetic fibres are of interest for several reasons. The enophthalmos, the narrowing of the palpebral fissure, the lowering of intra-ocular pressure, and the constrictor effect on the pupil (due to unopposed action of the parasympathetic fibres of the third nerve) follow whenever the preganglionic or the postganglionic fibres are divided. This means that the superior cervical ganglion and its fibres, when isolated from their preganglionic connections, exert no influence on the smooth muscle of the orbit. The peripheral ganglia are not centres from which reflex effects can be initiated. It is true that this question has been investigated often and much discussed, but the laboratory observations and the surgical results seem to be in agreement that the isolated peripheral sympathetic ganglia are inactive when severed from their spinal connections.

The ocular effects are continuous and have been observed for several years after removal of the stellate ganglion, i.e., by breaking their preganglionic connections. All sympathetic effects are not continuous and tonic. The ocular and vasoconstrictor belong to this group, and also perhaps the pilomotor and the sudomotor. Sweating, however, is somewhat complicated, for there is a sweating that depends on the spinal nerves, that is activated by pilocarpine and inhibited by atropine - a parasympathetic resemblance. It is well known that adrenalin, the normal sympathomimetic drug of the body, does not activate the sweat glands. The effects on the unstriated muscle and glands of the alimentary system would appear to be occasional and not of a continuous tonic nature. This distinction is of importance, for obviously surgical intervention is likely to be more effective when it is directed to the removal of the continuous tonic effects.

The anatomical pathway of the distribution illustrates another aspect of the peripheral sympathetic system. The nerve-fibres reach their destination by travelling with the blood-vessels. This would seem to be the usual way in the case of the viscera. For the visceral structures we have long preganglionic pathways - the cervical sympathetic pathway in part, the great splanchnic, and the hypogastric nerves (the presacral). These encounter peripheral ganglia: the superior cervical ganglion, the celiac and the inferior mesenteric ganglion, together with the cells in the superior hypogastric plexus. From these ganglia then proceed the final fibres, and these reach their destination by travelling with the blood-vessels. This is in marked contrast with the distribution to the parietes and limbs, where the fibres can be observed as grey rami joining the segmental spinal nerves.

The evidence from morphology and function set out above induces us to suggest that these two modes of distribution represent a real distinction in the arrangement of the sympathetic nervous system, and that each part, the somatic and the visceral, preserves its characteristic mode. The ocular effects are so certain and easily recognizable that they can be used, if necessary, as a criterion for the anatomical success of a cervical sympathectomy. The presence or absence of Horner's syndrome makes it certain whether the cervical sympathetic has been divided and the stellate ganglion removed or not.

We have referred to the restriction of the spinal-cord area from which the preganglionic fibres to the peripheral ganglia start. Mention has been made of the fact that each preganglionic fibre subserves several peripheral ganglia. Further, if we inquire into the higher centres which activate the lower spinal centres we should find them congregated in a small area in the hypothalamus. Thus we are prepared for the reception of the notion that a third and possibly the most important activity of the sympathetic system is its total emergency response. This aspect, though of the greatest importance, is not germane to our present intentions, for it is outside the scope of surgical intervention. We mention it now to invite attention to the threefold quality of sympathetic activity: the continuous tonic aspect, which offers reasonable hope that surgical intervention may be profitable; the occasional, such as the effects on the gut, where the results of surgical intervention are perhaps less likely to be reliable; and the total emergency response, which is outside the scope of surgery. A piecemeal intervention is not likely to hurt this last response, for the peripheral-fibre system is helped out by the production of adrenalin.

Two deductions from this are of further surgical interest—namely, the anatomy of the system and the results of physiological investigation do not suggest that local diseases are likely to find their explanation in hyper-activity or hypo-activity of small parts of the peripheral system. The effect most likely to be achieved by surgery is an indirect one—namely, an increase in the amount of blood flowing to a particular region through the removal of vasoconstrictor tonic impulses. Tonic impulses to smooth muscles, either activatory or inhibitory, could be removed, but beyond such effects as these there is no evidence at the moment that more can be achieved.

THE SYMPATHETIC INNERVATION OF THE UPPER LIMB.

The sympathetic innervation of the upper limb leads us to examine in the first instance the origin of the rami and their connection with the spinal nerves that make up the brachial plexus.

It is possible for a body structure to receive sympathetic fibres by three routes: (1) By incorporation with the peripheral spinal nerves and thus sharing in their distribution; (2) By proceeding with the blood-vessels; (3) By direct distribution to the organ without joining other kinds of nerves or accompanying blood-vessels. The last way arises in the case of some of the abdominal and pelvic viscera. In the limb, only the two former arise.

The Vascular Route.—The opinion that the vascular route is used by sympathetic fibres has diffused widely following the observations and surgical reports of Leriche. Actual observation discloses, of course, the penetration of the spinal nerves by the rami; it shows fibres from peripheral nerves joining blood-vessels, which in accordance with ordinary dissecting-room procedure may fairly be called branches of vascular distribution. Numerous nerve-fibres, in the form of fasciculi, running for considerable distances, can also be observed in the adventitia of blood-vessels; and finally in the tunica media

a continuous nerve plexus can be seen extending throughout the whole of the vascular tree. Moreover, by the ordinary method of dissection aided by a binocular, it is possible to trace some fibres for a short distance on stem-vessels like the subclavian and the iliac arteries. The fewness of the fibres and the difficulty of the dissection here is in marked contrast with the ease of tracing such nerves on the carotid or splanchnic vessels, for instance.

The method of dissection by exposing the rami and the branches of vascular distribution from adjacent nerves suggests strongly that in the limbs the sympathetic fibres reach their destination through the spinal nerves. The investigations of Langley, Woollard, and others have brought experimental confirmation of this, and we believe that surgical opinion is now in alignment with this view.

Perhaps the simplest and most convincing demonstration of the fact that the blood-vessels of an extremity get their innervation from the sympathetic fibres in the adjacent nerves is to inject a peripheral nerve, such as the median or ulnar, with novocain. Along with the anæsthesia a paralysis of the sympathetic fibres occurs, and the removal of constrictor impulses discloses itself in vasodilatation, producing a flush and a rise of temperature. In this way it can easily be shown that the vasodilatation coincides with the peripheral distribution of these nerves, and in the case of the ulnar a difference of temperature can be shown, by using a thermo-couple, between the two sides of the ring finger (Woollard and Phillips). The method of cocainization of peripheral nerves, or of spinal anæsthesia, or the production of protein reaction (typhoid vaccine), has been used as a clinical test to determine the part which sympathetic impulses are playing in the production of particular lesions.

The converse method of infiltrating the adventitia of blood-vessels like the radial artery gives no vasodilator response. It can be concluded with certainty that the experimental, the degenerative, and the dissecting methods all agree in deciding that the blood-vessels of a limb receive their sympathetic fibres by distribution from adjacent nerves.

Since, however, there is demonstrable a continuous plexus in the media of blood-vessels, it might be supposed that this could afford a chance for impulses to proceed upwards or downwards within it. This plexus is unlike a conducting system. It gives off pericellular endings to the unstriated muscle fibres. Impulses which enter this plexus extend over a small area and then die out. Though its appearance suggests continuity, yet the arrangement is of such a nature that its purpose is to subserve a restricted portion of the vascular tree, and experiment shows that impulses soon die out within this area and are not transmitted for any distance upwards or downwards.

The nature of the intrinsic plexuses is of some importance, for we encounter a similar arrangement in the ureter. This will be mentioned again later. But it is necessary to emphasize at the moment that organs and blood-vessels have no break in the continuity of their intrinsic plexuses, and that these function locally, while from the periphery come the fibres whose subdivisions form these plexuses.

THE ANATOMY OF THE RAMI AND THE GANGLIA.

The number, arrangement, and apparent source of the rami, as well as the cervical sympathetic ganglia themselves, exhibit considerable variation. Potts, Axford, and others have recorded from their dissections variations in the number of rami, and the inconstancy of their relations to adjacent muscles. Our own observations are in agreement with the results they reached. The feasibility of such operations as ramisection must be determined in large part by the assortment of these sympathetic branches, and also the variations encountered raise the problem whether any significance attaches to the number of these rami.

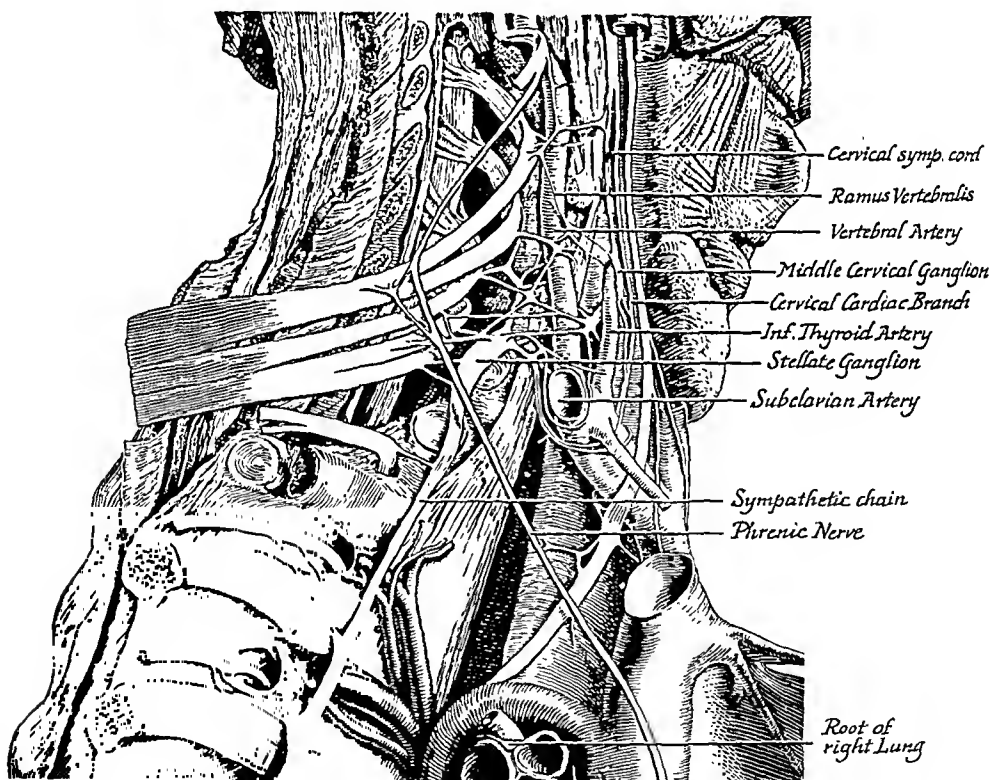


FIG. 66.—Dissection showing the connections of the lower cervical sympathetic system.

The teaching commonly imparted of the anatomy of the cervical sympathetic would lead one to suppose that the inferior cervical ganglion and the first thoracic ganglion are discrete masses, but such would seem rarely to be the case. Most often it is found that an elongated ovoid mass is situated in front of the neck of the first rib near the costocentral articulation, and that this mass, when traced upwards to the lateral angle between the vertebral artery and its origin from the subclavian, emits a stream of fibres in which one or more small ganglionic masses are entangled. It is an economy to

apply the term 'stellate ganglion' to all these ganglionic accumulations rather than to attempt to delimit one part as first thoracic, another as inferior cervical, and another as the middle cervical ganglion.

By references to the accompanying figures (*Figs. 66, 67*), it can readily be apprehended that the ganglionic masses, in order to be exposed, need division of the *sealenus anticus* muscle, displacement of the subclavian artery, and the separation of the extrapleural tissues from the medial border of the

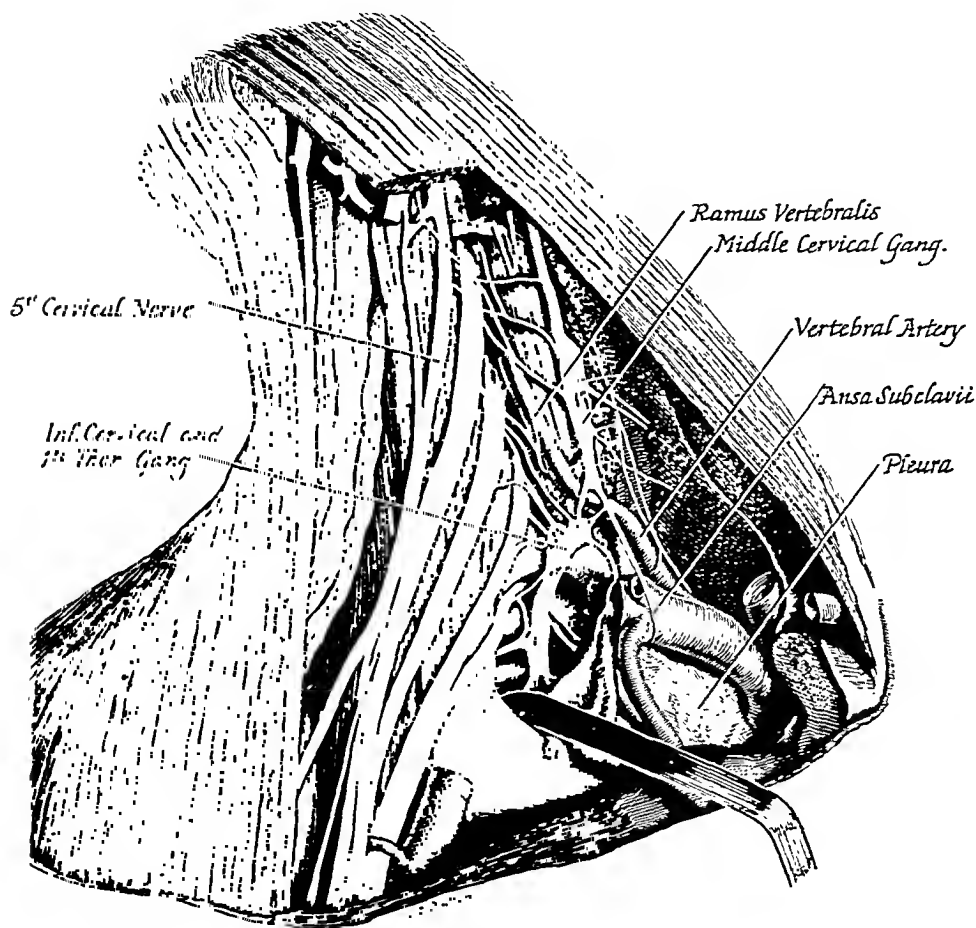


FIG. 67.—A dissection showing the positions and connections of the middle cervical and stellate ganglia.

first rib. The lower pole of the ganglionic mass leads directly downwards and backwards to the second thoracic ganglion, which lies opposite the medial extremity of the first intercostal space and usually extends to the upper border of the second rib.

The middle cervical ganglion is a very uncertain topographical mass. It is usually assigned to a place where the cervical sympathetic chain crosses

the inferior thyroid artery opposite the sixth cervical vertebra. Reference to the same figures (*Figs. 66, 67*) will show that in one case (*Fig. 66*) the mass labelled 'middle cervical ganglion' is an inconspicuous thickening on the cervical chain, while in the second case (*Fig. 67*) it forms a mass easily divisible into upper and lower portions. Since the middle cervical ganglion is such a fluctuating quantity, similar variation must occur in the origin and course of the rami to the fifth and sixth cervical nerves which are usually attributed to it.

If the term 'ganglion stellatum' were extended to cover all the large and small ganglionic masses opposite the neck of the first rib and extending upwards into the angles formed by the origin of the vertebral and inferior thyroid arteries from the subclavian, it would get rid of some artificialities in descriptive anatomy and would make the use of the term approximate more nearly to its connotation in comparative anatomy. The irregularities of these ganglionic masses remind us more of the cœliac plexus than of segmental ganglia. The attempt to emphasize this latter aspect has led to the terms usually employed. They are of course also the source of visceral fibres (most, if not all, of the cardiac accelerators arise at this level) as well as of segmental rami.

The rami take origin from the main ganglionic mass for the most part, but some come as well from the more cranially lying portions. It would be tedious to describe in detail their plexiform arrangement, their varying relations to the adjacent muscles, their relations to the arteries, and the inconstancy in their number and apparent origin. Excellent descriptions have been furnished by Axford and others. It will suffice if we describe the results of one of our dissections.

In this specimen (*Fig. 67*) the inferior ganglionic masses in the cervico-thoracic region consist of an upper, middle, and lower mass. The term 'middle cervical ganglion' would be appropriate to the upper two on topographical grounds. The connecting links between these lie behind the subclavian artery and encircle the vertebral artery. From the upper mass a single ramus joins both the fifth and the sixth cervical roots. A medial visceral ramus is also present. From the middle mass a large ramus proceeds upwards and outwards to the fourth and fifth cervical roots. Two medially directed visceral branches are also present. From the connecting fibres between the middle and lower ganglionic masses a ramus joins the seventh cervical nerve and the ramus vertebralis proceeds with the vertebral artery. The ramus vertebralis is variable in size but always present, and furnishes an additional route by which rami join the upper cervical roots. These enter the cervical roots independently of those which come more superficially from the ganglia. The lowest ganglionic collection supplies a small ramus to the sixth cervical, and a larger ramus is distributed to the sixth, seventh, and eighth cervical nerves. A separate small ramus proceeds to the first thoracic nerve. In addition there are, of course, visceral branches.

Kuntz has drawn attention to the occasional presence of an additional ramus to the first thoracic nerve from the second thoracic ganglion. This ramus we have encountered on one occasion. Its frequency is great enough to impose always on the surgeon the need for designing his cervical sympathetomy operations in such a way as to destroy this ramus.

It is clear from this compendium that the rami are more numerous and more widespread than might have been supposed. Further, they have a widespread origin from a stellate ganglion or from the more separate masses (first thoracic ganglion and inferior cervical ganglion), from the cervical sympathetic at, below, or above the supposed position of the middle cervical ganglion, and from the ramus vertebralis. Again, rami may be separate at their origin and conjoin later, or, on the other hand, a ramus of single origin divides and so supplies more than one spinal nerve.

These several rami enter into the spinal nerves over varying intervals, and may pass in front or through the prevertebral muscles and the scalenus anticus. Thus, on anatomical grounds it would be hard to believe that the operation of ramisection, i.e., the cutting off of the sympathetic fibres to the limb by division of the rami, would ever be complete. There are, of course, other reasons for directing the surgical intervention elsewhere.

We have so far proceeded on the assumption that all these rami are postganglionic, i.e., that they have come from the cord not higher than the first thoracic ventral root and have their cell stations in the ganglia above described. This piece of anatomy rests, we believe, on a quite secure basis, but there are not wanting observers who would elevate the level in the spinal cord from which such fibres might come. Winkler, for instance, on histological grounds, has suggested that such fibres might reach the upper part of the cervical sympathetic by way of the spinal accessory. This is highly improbable, and a re-examination of the ventral roots for finely medullated fibres brought us to the same conclusion as Harman long ago reached—namely, that ordinarily the first thoracic ventral root is the first root in which such fibres become conspicuous. This morphological test by itself is not conclusive, for finely medullated fibres which are not sympathetic but motor in function exist in all ventral roots (Sherrington). These are few and scattered, however, and the morphological picture of the first thoracic root contrasts strongly with those preceding it.

Harman drew attention to the fact that the sympathetic outflow does vary, however, with the fixation of the plexus. If this moves forwards a segment, then the preganglionic outflow shifts to the eighth cervical segment, or if post-fixed the second thoracic root becomes the most cranial level of the outflow. This, we think, explains the occasional contribution from the second thoracic ganglion to the first dorsal nerve, to which Kuntz has directed attention. The number of rami exposed by a dissection is not by any means a measure of the number of sympathetic fibres joining the spinal cord. Some of the rami really consist of a small blood-vessel and a few fibres. The usual method of dissection is likely to be complicated by the presence of more or less fibrous tissue, and, indeed, a fibrous strand may be so dissected as to be mistaken for a nerve-fibre. Histological investigation is necessary as well, in order to determine the true composition of the rami exposed by dissection. This does reveal that some of the rami found by dissection may owe their apparent size to fibrous tissue or to the presence of a small blood-vessel and contain but few nerve-fibres. The nerve-fibres are almost entirely non-medullated, but in some rami there will be found a few fibres that have not yet lost their medullary sheaths. Despite the presence of medullation.

it is justifiable to assume that these are postganglionic fibres, for a similar state has been noted in other parts of the body in fibres definitely known to be postganglionic.

As has already been mentioned, the evidence justifies us in believing that these rami undergo along with the other nerve components of the brachial plexus the same reassortment and arrangement, and thus have the same distribution.

Since also it is known that the vasomotor supply is most abundant to the more superficial vessels, and, of course, the pilomotor and sudomotor are entirely cutaneous, we might infer, therefore, that those spinal segments which innervate the largest cutaneous areas would contain the greatest number of sympathetic fibres. Actual observations suggest that the largest number enter the eighth cervical root in the brachial plexus and the first sacral in the lumbo-sacral plexus, i.e., to the hand and foot regions.

Though ramisection would be justified on physiological grounds, the anatomical difficulties of achieving a sympathetic denervation in this way are, as already pointed out, very considerable. There is, however, another reason why ramisection is unsuitable. The vascular changes and the ocular effects which follow sympathectomy have now been observed for a long time after operation, and Adson and Hesse have noted that they are present for two to three years. This means practically that they endure indefinitely. This would, of course, not be so if regeneration could occur. Enough clinical evidence is now available to make it likely that such regeneration does occur after ramisection and may be practically complete in a year or so.

From these observations and the experience now available, it can be concluded that the only way to denervate the upper limb of its sympathetic fibres, to avoid all regeneration, and to ensure a permanent state of relative vasodilatation, is to remove those masses comprised in the term 'ganglion stellatum', i.e., the inferior cervical ganglion and the first thoracic ganglion of ordinary anatomy. Since the second thoracic ganglion occasionally makes a contribution to the sympathetic innervation of the upper limb, this should be included also. The problem of the most advantageous approach, from the front or the back, we do not propose to discuss, since other than anatomical considerations arise. We can assert, however, that the anterior approach has been completely successful on many occasions in the Surgical Unit of St. Bartholomew's Hospital, and subsequent dissection of the excised tissue has proved that the second thoracic ganglion has been removed.

THE SYMPATHETIC INNERVATION OF THE DISTAL PART OF THE COLON.

The mode of sympathetic innervation of the gut stands in sharp contrast to that of the limbs. For in the alimentary tract the ganglia from which the peripheral fibres start are piled up along the front of the aorta. These ganglia are fed by preganglionic nerves which travel relatively a very long distance before they reach their synaptic terminations. The peripheral fibres run in close association with the blood-vessels in order to reach their destination. The nodal ganglionic point in the case of the descending colon, the

iliae colon, the pelvic colon, and the rectum is the inferior mesenteric ganglion. This perhaps needs some qualification in the case of the rectum and distal part of the pelvic colon, for there occurs here a slender reinforcement of sympathetic filaments derived from the hypogastric and pelvic plexus. The inferior mesenteric ganglion is, however, not a discrete mass, and its scattered elements may have an extensive range. They may be found proximal to as well as at the level and distal to the origin of the inferior mesenteric artery. Scattered cells are also found in the hypogastric plexus. The fibres coming to this ganglion are derived in chief part from filaments which descend along the anterior surface of the aorta, having passed through the cœliac plexus. In their descent they form a pre-aortic plexus. In addition, branches are given to the inferior mesenteric plexus from the second and third lumbar ganglia on each side. These pass forwards and downwards behind the beginning of the inferior mesenteric artery to mingle with the other fibres of the inferior mesenteric ganglion. The inferior mesenteric plexus thus constituted then subdivides so that each branch of the inferior mesenteric artery is accompanied by several large filaments. Those which accompany the superior hæmorrhoidal artery cross the common iliac vessels and eventually form a plexus behind the rectum in a plane anterior to that of the hypogastric nerves. These two plexuses communicate a few fibres running between the superior hæmorrhoidal plexus and the hypogastric plexus. To deprive those portions of the gut which lie within the vascular bed of the inferior mesenteric artery of their sympathetic nerve supply, it would therefore be necessary to remove the inferior mesenteric ganglion. To render the denervation as certain as possible, the dissection should be extensive and should be made proximal to the origin of the artery by removing the adventitia from the front of the aorta. In order to secure the contributions from the lumbar ganglia, the inferior mesenteric artery should be stripped of its adventitia for a short distance from its origin. Since the hypogastric plexus contributes some fibres to the superior hæmorrhoidal plexus, this also should be included in the resection.

The anatomical considerations are not entirely ended by breaking the sympathetic fibres at the level of the inferior mesenteric ganglion. As is well known, there exists in the gut the intrinsic plexuses of Auerbach and Meissner. The anatomical relationship of these intrinsic plexuses to the extrinsic nerves of the gut, such as the vagal and sacral parasympathetic and the sympathetic nerves just described, is, in our opinion, quite unsettled. Without opening up the whole question in these pages, we would content ourselves by asserting that there is at the present time no justification for removing it from the separate category in which Langley placed it—namely, that portion of the autonomic nervous system which he called the 'enteric nervous system'. The matter is important, for it affects the view we must take in regard to the motility and vasomotor reactions of the gut. There is, of course, abundant evidence of the presence of vasoconstrictors in the gut, but there is also evidence of vasodilator fibres as well. In regard to the gut movements, opinion has crystallized around the conception that the segmental movements are myogenic in origin, the peristaltic are due to the intrinsic nervous plexuses, and the extrinsic systems, like the sympathetic and parasympathetic.

are regulatory in function. Thus the sympathetic, when stimulated, inhibits the peristaltic movement of the gut, and, almost as a necessary concomitant of this, enhances the tonic activity of the sphincters. The pathological evidence gained from the study of the achaliasias, though as yet we think not conclusive nor yet analysable into cause and effect, does suggest that degeneration of the intrinsic plexuses is associated with these. In any case, whatever be the interpretation of these changes in the intrinsic plexuses, they are outside the range of surgical intervention, and so the soundness of operations on the sympathetic nervous system for these disorders of gut motility must depend on clinical observations.

INNERVATION OF THE BLADDER AND URETER.

Although the more important anatomical features of the innervation of the bladder are known, there are still some points which need further elucidation. The principal sources of the nerves of the bladder are by way of the hypogastric (presacral) nerves and plexuses and by way of the sacral parasympathetic nerves (pelvic nerves). The pudic nerve is the sensory nerve of the urethra and the motor nerve of the external sphincter of the bladder. Since the external sphincter of the bladder, especially in women, contains a considerable number of unstriated muscle fibres, there must be some overlap in the motor elements of the three nerves supplying the bladder. The sacral parasympathetic nerves to the bladder contain the most important afferent and efferent nerves that have to do with micturition, and therefore cannot be touched by the surgeon. In those cases where it would appear that the difficulty in micturition is due to the inability to relax the external sphincter, some relief might be expected to follow a partial resection (avoiding the branches to the anal sphincter) of the pudic nerve.

At the moment surgical intervention is directed most usefully to the sympathetic fibres that reach the pelvic viscera by way of the hypogastric nerve. It is necessary to consider, in view of the anatomical results of other investigators, if this is the only route by which such fibres travel. Another source which has been mentioned is by fibres passing directly to the bladder and other viscera of the pelvis from the chain of sacral sympathetic ganglia. Our own dissections have been especially directed to this point and occasionally we have come across such connections, but they are not constant. When they are present they are extremely small. We have doubted whether such connections are real in the sense of containing nerve-fibres, but histological examination has proved that they contain a few. We are of opinion that this source of fibres is inconstant, that at most only a few fibres pass by this route, and that it may well be that these are used entirely for the innervation of adjacent small blood-vessels.

Observations on the Formation and Distribution of the Hypogastric Nerves.—Another route has been postulated, chiefly by French surgeons—namely, that sympathetic fibres reach the pelvic viscera by travelling along the blood-vessels. We have already remarked on the fact that when this route is the channel for the peripheral distribution such a route is easily identified by the ordinary method of dissection (*Fig. 68*), which shows the

nerve-fibres accompanying the superior hæmorrhoidal artery. When such fibres are not so seen we are really confronted by the same problem which has been raised and solved in the case of the limbs. The continuous innerva-

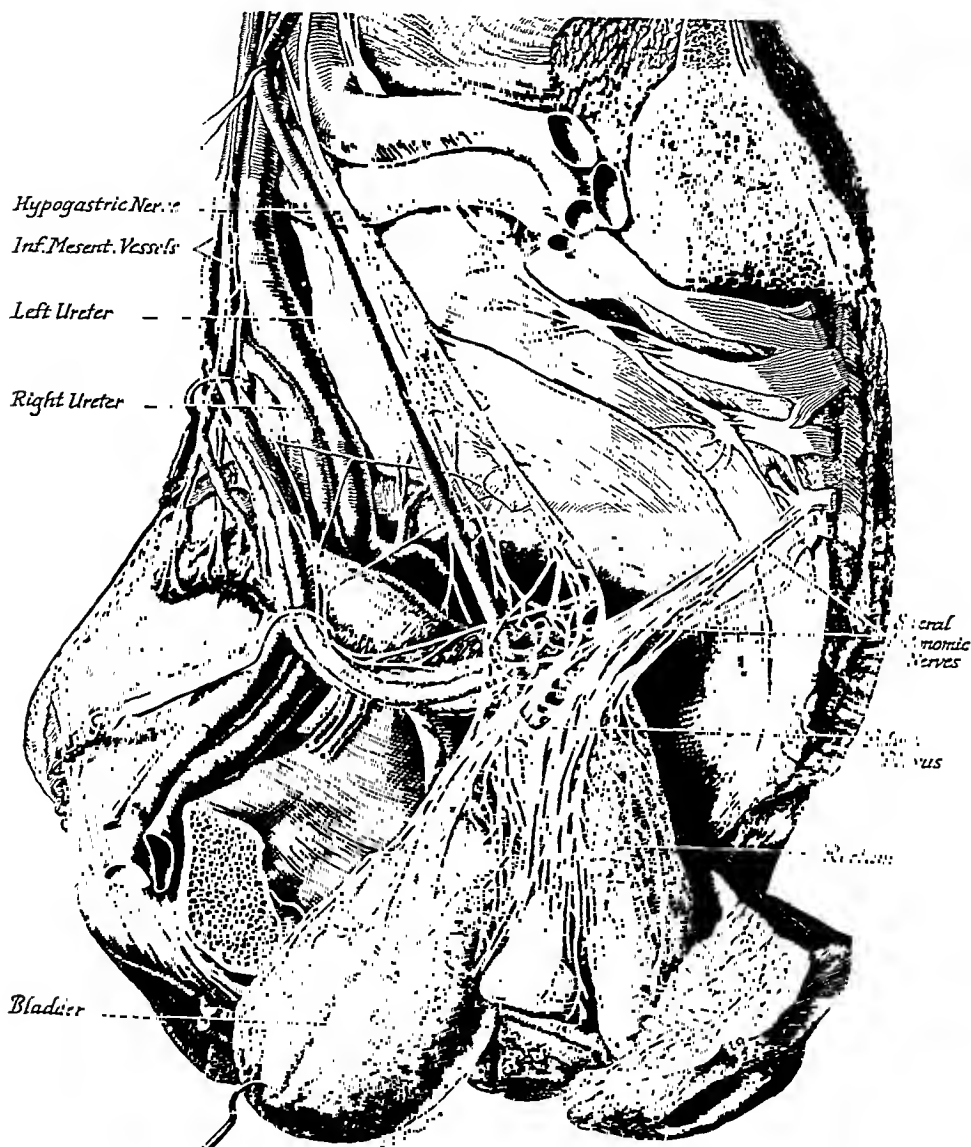


FIG. 68.—A dissection showing the distribution of the hypogastric and pelvic nerves.

tion by an intrinsic nerve net in the blood-vessel is the mode of local innervation and not the method of transmitting impulses to a distance either in the blood-vessels themselves or to the structures which they supply. Thus we

believe that practical surgery is best served at the moment by a knowledge of the anatomy and physiology of the hypogastric nerves.

These hypogastric nerves are believed to be inhibitory to the detrusor of the bladder and tonie to the so-called internal sphincter of the bladder. One uses the qualifying phrase 'so-called' because anatomical investigation in this department throws some doubt on the usual conception of such a sphincter. However, this does not affect the way these nerves co-operate in determining the action of the bladder. Both animal experiment and surgical intervention go to prove that this contribution of the hypogastric nerves to the function of the bladder is not one which need prohibit the intervention of the surgeon. A slight increase in frequency of micturition is the usual result of its removal and even this is probably temporary. Indeed, this result is an indication for resection of this nerve, for there occur cases of difficulty of starting the action of emptying the bladder which appear to be due to too much inhibition through the hypogastric.

These same nerves are also vasoconstrictive, and this action emerges as the opposite function of the sacral parasympathetic ('pelvic nerves' of some authors, also called 'nervi erigentes'). Further, they are motor nerves to the vesiculæ seminales and prostatic gland. Thus after their exclusion the contribution of these organs by which the seminal fluid is formed is absent, and so the male may be sterile though not impotent.

There is evidence that afferent fibres reach the central nervous system by passing through the hypogastric nerves. This evidence has been obtained from the study of spinal cases in man. Riddoch and Head observed patients in whom, through spinal injury, all nerves to the bladder except the hypogastric nerves were cut off from the higher centre, and found that they were aware of the state of the distension of the bladder. Learmonth divided the hypogastric nerve in man, using local anæsthesia, and stimulated the central end. The patient complained of a pain in his bladder as though it were being crushed. Histological investigation shows the presence of large medullated nerves, 12 to 15 μ in diameter. In the light of the foregoing evidence and what we know of the anatomy of sympathetic fibres it is not unreasonable to believe that these large medullated fibres are sensory in function.

The Anatomy of the Hypogastric Nerves (Presacral Nerves).—In view of the foregoing comments on the anatomy, we present in some detail the results of our dissections.

The hypogastric nerves vary considerably in arrangement and form, and the following observations have been derived from the study of six cadavers. Despite the individual variation, the general arrangement presents a fairly constant pattern. In three specimens (*see Fig. 68*) dissected by us the nerve consists of a single strand 2 to 4 mm. in breadth lying in the mid-line across the promontory of the sacrum. It is formed by the union of two nerves of approximately equal size at the level of the left common iliac vein, and lies in a separate fascial stratum in front of the middle sacral artery. It is surrounded by a variable amount of fat. Below, the nerve spreads out into a plexiform network of fibres placed over the first sacral vertebra and upper part of the second. Above, the two roots are derived from a plexus situated on the front of the aorta. The root of the right side receives fibres from the

second and third lumbar ganglia and from the pre-aortic plexus. The strands arriving from the right ganglia are seen to pass behind the inferior vena cava on to the front of the aorta. The root of the left side is formed in part from the inferior mesenteric ganglion and in part from the branches derived from the second and third lumbar ganglia, these latter passing behind the origin of the inferior mesenteric artery in order to reach the left hypogastric nerve. The right and left nerves so formed then cross the common iliac artery of

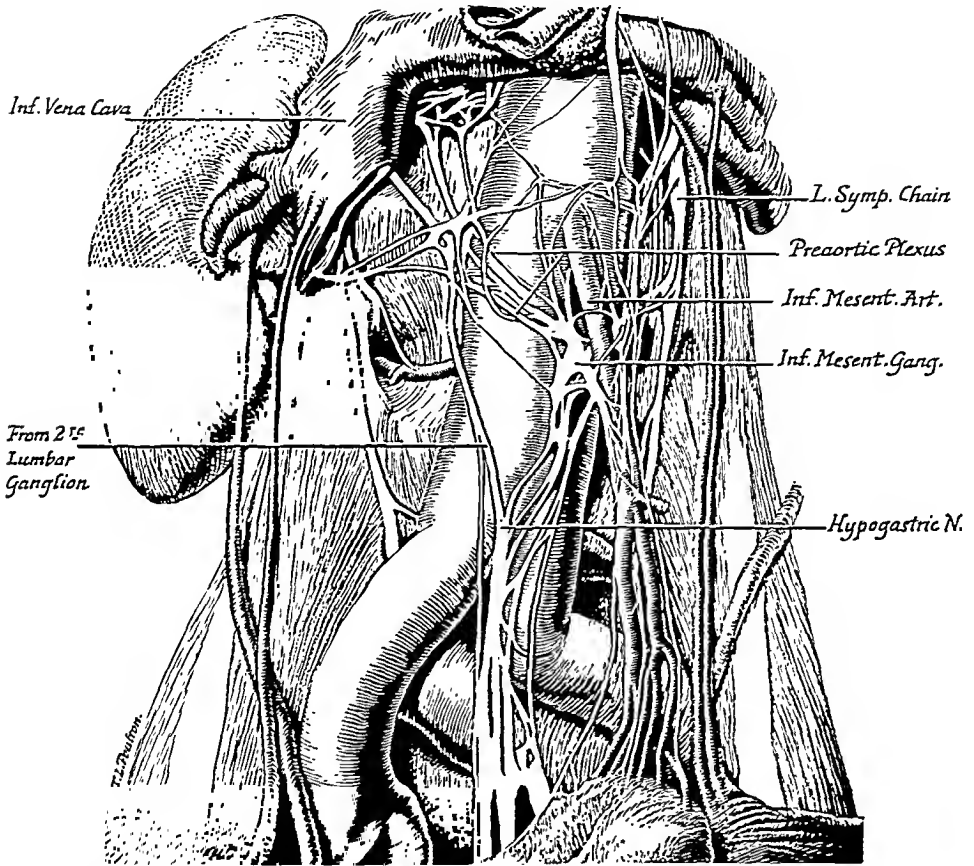


FIG. 69.—A dissection showing the position and connections of the inferior mesenteric ganglion and the formation of the hypogastric nerves.

their respective sides and join at or near the lower border of the left common iliac vein.

In one of our specimens (*Fig. 69*) the hypogastric nerve was present as a broad band placed on the left common vein and formed by the coalescence of seven different contributions. One of these, the largest, descended on the right side of the aorta from the pre-aortic plexus. It was joined by six smaller branches which crossed together the left common iliac artery from the inferior mesenteric ganglion. This group represents the simpler left

single radicle in the previous specimens. Almost directly after its formation the hypogastric nerve breaks up into a plexus in front of the first piece of the sacrum.

Intermediate in type between these were two specimens showing the formation of the two main roots which form the hypogastric or presacral nerves from a lesser number of contributory fibres. In one a branch of fairly large size passed on the left side behind the left common iliac artery from the inferior mesenteric ganglion to join the plexus resulting from the subdivision of the main nerve. This branch might easily escape the surgeon. In summary it may be said: (1) That the hypogastric nerve is derived from two main roots. (2) These two roots vary from a single strand to a group of fibres crossing either the bifurcation of the aorta or the iliac arteries of each side. (3) The right root comes mainly from the aortic plexus and receives fibres from the second and third lumbar ganglia of that side. (4) The left root comes partly from the inferior mesenteric ganglion, partly from the aortic plexus, and partly from the second and third lumbar ganglion of that side. (5) One or more branches may pass behind the left common iliac artery. (6) The hypogastric nerve lies in a separate stratum of fascia and may be embedded in a considerable amount of fat. In one specimen only were fibres found passing from the fourth lumbar ganglion on each side.

The Pelvic Plexus.—The hypogastric plexus in front of the sacral vertebrae divides into two general divisions which pass laterally and forwards behind the peritoneum of the recto-vesical or recto-uterine pouch. In two specimens the nerves were found to be fairly close to the peritoneum. Reaching the region of the base of the bladder a series of fibres, six to ten in number, were found to turn up on the vesical end of the ureter and to run upwards along this structure. A dense interlacing plexus was formed at the side of the prostate and bladder base fed by the hypogastric passing downwards and laterally from above and by the sacral parasympathetic nerves from behind.

The sacral parasympathetic nerves come from the third and fourth sacral nerves; occasionally an additional rootlet comes from the second sacral nerve. These nerves are composed of six to eight considerable strands on each side, $1\frac{1}{2}$ in. in length, passing on either side of the rectum and in close proximity to it. They lie below the level of the reflection of the peritoneum on each side.

In two specimens only a fine filament was found connecting the sacral sympathetic ganglia to the plexus on each side. This came from the third sacral ganglion of the sympathetic and was the only connection found passing to the hypogastric and pelvic plexuses from the sacral sympathetic ganglia.

These dissections from a surgical point of view represent the anatomy of the hypogastric nerves and confirm so far as this method permits that a complete sympathetic denervation can be accomplished by removal of this system.

Some variation occurs in the arrangement, but it is an invariable rule that all the fibres forming this nerve become aggregated and plexiform over the fifth lumbar vertebra and promontory of the sacrum, and that if the peritoneum is reflected from the interval between the common iliacs, the

whole of this system—the superior hypogastric plexus of ordinary anatomy—can be removed.

Dissections of the material removed by several different surgeons from this region between the common iliacs show that it is possible to make such surgical exsection anatomically complete. It should be mentioned that Langley found by degeneration methods that a very few fibres of the pelvic nerves (0-4) reach the hypogastric plexus. These nerves are so few that it does not seem necessary to modify any of the previous statements.

The anatomical and physiological aspects of the hypogastric nerve set out above suggest that its removal may be easily accomplished. The removal is carried out at a point where some fibres are still preganglionic, but where most are postganglionic. Our investigations show that a tract of ganglion cells is included in this area. It begins above in the sometimes compact but more often diffuse inferior mesenteric ganglion, and the cells are continued in a more scattered manner in the superior hypogastric plexus. Thus regeneration will not occur.

The point we would make is that in so far as it may be deemed desirable to denervate the pelvic viscera of their sympathetic fibres, this can be accomplished by a retroperitoneal dissection over the fifth lumbar vertebra and promontory of the sacrum between the iliac vessels. Attempts at the removal of the sacral sympathetic ganglia, the lumbar sympathetic chain, or stripping the adventitia from the various blood-vessels entering or within the pelvis, have all been advocated. All these we believe to be unnecessary. The physiological effects of a pelvic sympathectomy can be obtained as stated above. Though we, of course, have been interested in the technique of this operation and the results obtained, we do not propose to discuss these. They, however, do include many cases of pelvic pain, especially in the female, some sexual disorders of the male, some cases of bladder pain and dysfunction.

The innervation of the ureter is in some ways analogous to the innervation of a blood-vessel, in others it resembles the innervation of the gut. A considerable literature has grown up around this matter. Its full discussion would be out of place here. We would present only the main result of our investigation. The ureter gets its nerves from the hypogastric sympathetic system and from the sacral parasympathetic system, and both these reach the ureter near where it enters the bladder. The ureter receives no nerve-fibres along its abdominal or pelvic course other than those which invest and reach it near where it enters the bladder. A plexus of nerves outside and within its muscular coat spreads up the ureter through the whole length of its course from this paravesical plexus.

This arrangement resembles what occurs in the blood-vessels, while the presence of a plexus of double origin with parasympathetic ganglion cells is, of course, an arrangement made familiar by what happens in the case of such viscera as the heart, etc.

The point which emerges from this statement of the anatomy is that, if it is deemed desirable to remove the influence of the sympathetic fibres from the ureter, this can be done by resection of the hypogastric plexus as described for the bladder.

THE SYMPATHETIC INNERVATION OF THE LOWER EXTREMITY.

The observations made on the upper extremity apply in the same degree to the lower extremity. Some degree of permanent vasodilatation most manifest in the foot can be set up by breaking the continuity of the sympathetic fibres to the limb. The most convenient place at which this can be done is in the lumbar sympathetic chain. These chains can easily be reached through the peritoneum, which is stripped back from the aorta. The left chain is beside the aorta, while the right is covered by the inferior vena cava and is easily reached by displacing this vessel inwards. It is sufficient to remove the third and fourth lumbar ganglia with the intervening chain. This operation differs from that in the upper limb, where the connection of preganglionic and postganglionic fibres is actually broken at the stellate ganglion. Thus the removal of the ganglion makes regeneration impossible. In the case of the lower limb the ganglion cells of the third or fourth segments are removed, but all the segments below this level keep their ganglia and postganglionic fibres, the abolition of their physiological effects being due to removing a segment of their preganglionic fibres. Theoretically it is conceivable that regeneration might occur, but it is not likely that the gap caused by the removal of the strip indicated will be bridged.

SUMMARY.

1. Evidence of a general nature has been presented indicating that the sympathetic nervous system is laid down in a way that suggests a particular conformation and a precise anatomy for each region of the body.

2. From a surgical point of view the sympathetic innervation of any particular region can be determined by the method of macroscopic dissection. Innervating fibres that cannot be determined by this method are not of surgical moment.

3. Groups of structures with a certain anatomical homogeneity have a common source of supply of sympathetic fibres, and these have in each case a uniform way of reaching their final distribution.

4. Some physiological conclusions are discussed, and at the moment the most constant and useful result that can certainly be achieved is an increase in the blood-supply to the denervated member.

5. In such anatomical areas a nodal point can be chosen where most conveniently by surgical methods the sympathetic innervation can be broken.

In the case of the head and neck and upper extremity, this interruption is best achieved by removal of the sympathetic chain from the level of the second rib upwards as far as the lateral angle between the vertebral artery and subclavian.

For the large gut, within the distribution of the inferior mesenteric artery, the sympathetic denervation can be achieved by stripping the adventitia widely from the aorta, beginning above the origin of the vessel, going distal to its origin, and continuing on the vessel itself as far as possible, i.e., as far as its first branches. It is, in addition desirable to remove the hypogastric plexus as well.

In the case of the pelvic viscera, including the ureter, a sympathetic denervation can be accomplished by removing the hypogastric plexus.

For the lower extremity, a sympathetic denervation can be most conveniently accomplished by removing the third and fourth lumbar ganglia and the intervening chain.

The work that this paper is based on, and the surgical experience which lies behind it, have been made possible by our association with the Surgical Unit of St. Bartholomew's Hospital Medical College. We wish to thank Professor Gask, the Director of the Unit, who suggested the scope of this contribution and took such a lively interest in its progress.

BIBLIOGRAPHY.

- ADSON, A. W., and BROWN, G. E., "The Successful Treatment of Raynaud's Disease of the Upper Extremities by Resection of the Sympathetic, Cervico-thoracic, and Second Thoracic Ganglia and the Intervening Trunk", *Jour. Amer. Med. Assoc.*, 1929, xcii, 444.
- AXFORD, M., "Some Observations on the Cervical Sympathetic in Man," *Jour. of Anat.*, 1927, lxi, 301.
- ECCLES, J. C., and SHERRINGTON, C. S., "Numbers and Contraction Values of Individual Motor Units examined in some Muscles of the Limb" *Proc. Roy. Soc.*, 1931, B, 107, 535.
- ELLIOTT, T. R., "The Innervation of the Adrenal Glands", *Jour. of Physiol.*, 1913, xlv, 285.
- GASKELL, W. H., "On the Structure, Distribution, and Function of the Nerves which Innervate the Vascular and Visceral Systems", *Jour. of Physiol.*, 1886, vii, 1.
- HARMAN, BISHOP, "The Anterior Limit of the Cervico-thoracic Visceral Efferent Nerves in Man", *Jour. of Anat. and Physiol.*, 1899, xxxiv, 356.
- HESSE, E., *Die Chirurgie des vegetativen Nervensystems*, 1930. Staatsverlag Moskau, Leningrad.
- KONDRATJEW, N., "Zur Lehre von der Innervation der Bauch- und Becken-höhleorgans beim Menschen", *Zeits. f. Anat. u. Entwicklung*, 1929, xc, 178.
- KUNTZ, A., "Distribution of the Sympathetic Rami to the Brachial Plexus, its Relation to Sympathectomy affecting the Upper Extremity", *Arch. of Surg.*, 1927, xv, 871.
- LANGLEY, J. N., "Observations on the Medullated Fibres of the Sympathetic System and chiefly on those of the Grey Rami Communicantes", *Jour. of Physiol.*, 1896, xx, 55.
- LANGLEY, J. N., and ANDERSON, J. K., "The Constituents of the Hypogastric Nerves", *Jour. of Physiol.*, 1894, xvii, 177; "The Innervation of the Pelvic and Adjoining Viscera", *Ibid.*, 1895, xix, 71 and 372.
- LEARMONTH, J. R., "A Contribution to the Neurophysiology of the Bladder in Man", *Brain*, 1931, liv, 147.
- LERICHE, R., "De la causalgie envisagée comme une névrite du sympathique et de son traitement par la dénudation et l'excision des pleins nerveux périartériels", *Presse méd.*, 1916, April 20.
- POTTS, T. K., "Peripheral Connections of the Human Sympathetic Nervous System", *Jour. of Anat.*, 1925, lix, Pt. 2, 129-135.
- RIDDIOCH, H., and HEAD, H., "The Automatic Bladder, Excessive Sweating, and other Reflex Conditions in Gross Injuries of the Spinal Cord", *Brain*, 1917, xl, 188.
- ROSENBLUTH, A., and CANNON, W. B., "Studies on Conditions of Activity in Endocrine Organs. XXVIII. Some Effects of Sympathin on the Nictitating Membrane", *Amer. Jour. of Physiol.*, 1932, xcix, 398.
- WINKLER, C., *Opera Omnia*, 1918, xvi, 175. Haarlem: Bohn.
- WOOLLARD, H. H., "The Innervation of Blood-vessels", *Heart*, 1926, xiii, 319.
- WOOLLARD, H. H., and PHILLIPS, R., "The Distribution of the Sympathetic Fibres in the Extremities", *Jour. of Anat.*, 1932, lxxvii, 18.

MESENTERIC CYST AS A CAUSE OF INTESTINAL OBSTRUCTION.

By W. ARKLAY STEEL,

REDHILL HOSPITAL, EDGBURY.

THE occurrence of small cysts disseminated over the surface of the mesentery, omentum, and bowel wall is very uncommon. These cysts are placed most abundantly at the bowel margin. They may appear in a small cluster or they may range in large numbers over an extensive area of mesentery.

It is usually during a laparotomy for some entirely separate condition that their presence is casually noted. They may be examined and, inviting puncture, their clear, opaque, or straw-coloured contents are expressed as a watery, chylous, or a thin-jellied fluid. Such rupture would appear to be harmless, but obviously the number of the cysts may negative any consideration of this procedure, or of individual enucleation, being applied to them.

Very rarely indeed these cysts may assume such a prominence as to become the reason for operation, either in virtue of their size, with concomitant pressure embarrassment, or because of some complication in connection with the tumour. The following case therefore justifies its record as a rarity, associated as it is with features which are themselves exceedingly uncommon.

Vera B., aged 8, was admitted to hospital on March 1, 1932. Her parents, one brother, and two sisters were alive and well.

HISTORY.—The patient was normal at birth, and except for measles and troublesome enuresis she had had no illness of any importance. Although apparently healthy, she was reported as being 'difficult' and 'finicky with her food'. She frequently complained of vague pains in the abdomen. For the past year these pains had been becoming more frequent and pronounced. The pains were accompanied by vomiting. Her appetite was affected and she was unable to take food of any description for a day or two at a time.

The child had been ill for six days prior to her admission to hospital. Throughout this period she complained of abdominal pain which was very much more severe than in any previous attack she had experienced. The pain was spasmodic at first but later became more constant. Vomiting was profuse and frequent. There was no normal action of the bowels, and only after two enemata had been given was a small faecal result obtained.

ON EXAMINATION.—On admission the patient was extremely ill, apathetic, and obviously in pain (circumstances which rendered the interrogation and history both difficult and unreliable). The body was well nourished. The face was pale and at frequent intervals was grimaced with pain. Her breathing was hurried and grunting. The temperature was 99.2° F., pulse 112, respiration 30. Several of the teeth were carious. The tongue was moist and clean, while the fauces were normal. The abdomen was distended but moved freely with respiration. Palpation of the abdomen did not help

to localize the pain, which was vaguely and widely indicated by the child. Tenderness was generalized over the whole abdomen, while rigidity was present in the lower half of the rectus muscles. The abdomen was tympanitic to percussion. A rectal examination was made but nothing abnormal was found. The other systems on examination were found to be normal.

A diagnosis of intestinal obstruction, resulting from a plastic type of tuberculous peritonitis, was made. A plain X-ray of the abdomen (*Fig. 70*)



FIG. 70.—Plain radiogram showing marked distension of small intestine.

confirmed the diagnosis of obstruction, and owing to the child's grave condition an immediate operation was undertaken.

OPERATION.—Under spinal anaesthesia combined with gas and oxygen, the abdomen was opened by a right paramedian incision. Distended loops of small intestine were at once displayed. The seat of obstruction was found in the ileum about two feet from the caecum. Numerous cysts were present throughout the mesentery. One very large cyst had kinked and dragged its weight across the ileum leading to complete obstruction. The cyst and

mesentery, together with two or three inches of bowel on either side of the cyst, were resected and a lateral anastomosis was performed. The abdomen was closed without drainage. The patient made an uneventful recovery and was discharged on March 31.

PATHOLOGICAL REPORT.—On one aspect of the mesentery attached to the piece of bowel, at the attachment to the gut, there are three small thin-walled cysts, the largest being little more than 1 in. in diameter; these cysts are isolated and do not communicate in any way with one another or with the bowel. On the opposite side at the mesenteric attachment is a small cyst about 1 in. in diameter and by the side of it a large cyst about 3 in. in diameter. It proceeds from the mesenteric attachment of the mesentery to

the bowel, and this part of its wall is fairly thickened and passes irregularly on to the rest of the thin wall of the cyst.

The cyst hangs over the bowel and has become adherent to it at its side, so that at first sight the cyst appears to depend from the gut itself. The contents of the cyst, as of all others, were clear, pale yellow, and watery in consistence. The interior of the cyst showed no evidence that it was in any way multilocular. There was no connection of this cyst with any depression of the mucous membrane of the gut between the trabeculae of the muscular coats.

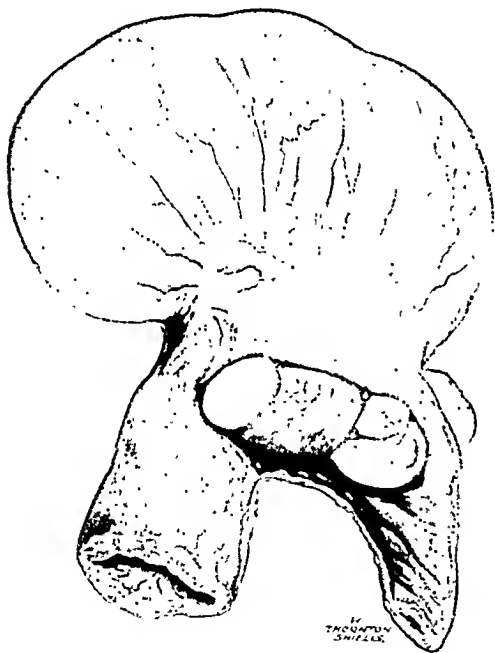


FIG. 71.—Mesenteric cysts completely occluding the lumen of the ileum.

X-RAY REPORT.—

Abdomen.—The plain radiogram shows five sausage-shaped translucent areas lying one above the other with their long axes transversely across the abdomen. The walls of these gas-filled areas are thin, and in addition fine close-set transverse striations are visible. The gas-filled areas measure 2 in. in diameter. A small translucent area below the left diaphragm is gas in the stomach.

X-ray Diagnosis.—Intestinal obstruction. "The close-set transverse striations indicate valvulae conniventes and not haustrations, and the distribution of the shadows is that of the small intestine. No cause seen for the obstruction."

The coloured figure (*Fig. 71*), which is an excellent reproduction of the appearance of the cyst and the resected tissue, shows the comparatively

large thin-walled cyst with its broad pedicle merging into the gut wall. Lying in the mesentery, bridging the loop of bowel, are smaller cysts; the one on the left slightly indenting the bowel and acting in counter-pressure to decrease the lumen of the ileum.

The actual site of obstruction is seen above this smaller cyst. The bowel is narrowed to the diameter of a pencil and is discoloured and gangrenous owing to the traction of growth and hanging weight of the large cyst. The proximal loop of ileum is seen congested and distended, while the distal loop is narrowed and collapsed.

The origin of mesenteric cysts is still a matter of controversy. The difference in their size and the disorderliness of their distribution, in relation to anatomical structures, in no way contributes a solution to the problem of their etiology.

As with most rare conditions, their pathology is not clearly understood, and is still further veiled by the general confusion of classification and nomenclature resulting from the changes and vagaries of opinion of those individual pathologists reporting on the few cases that come their way.

The cystic are more common than the solid mesenteric tumours, and are subject to numerous complications, of which intestinal obstruction is by far the most common and serious. Complications such as torsion, impaction, rupture of the cyst, or intracystic hæmorrhage are infrequently encountered.

Intestinal obstruction may arise in various ways, dependent on the size, position, and mobility of the cysts and their situation in the mesentery. The resulting acute obstruction is purely mechanical and is brought about by direct pressure or traction on the bowel, while the occlusion may be completed by adhesions or kinks, or, in very rare instances, by volvulus of the gut. The condition may be considered or suggested by the history, by isolating a mobile cyst, or by the pain, vomiting, diarrhœa, or constipation which arise from the pressure of the cyst. The diagnosis even in uncomplicated cases is notoriously difficult, but in the presence of intestinal obstruction an accurate diagnosis is practically impossible.

I am very much indebted to Dr. Geo. Simon for the assistance given me by his valuable and accurate opinion and for the radiogram reproduced here. I am further indebted to Dr. Teale for furnishing me with the pathological report, and to Mr. Thornton Shiells for the very accurate representation of this rare specimen. The specimen is now in the Museum of the University College Hospital.

BIBLIOGRAPHY.

- KENDALL, E., *Brit. Jour. Surg.*, 1930, xviii, 168.
 FLYNN, C. W., *Ann. of Surg.*, 1930, xci, 505.
 SWARTLEY, W. B., *Ibid.*, 1927, lxxxv, 886.
 PETERSON, E. W., *Amer. Jour. Surg.*, 1928, v, 514.
 GREEN, J. A., *Surg. Gynecol. and Obst.*, xlv, 401.
 CUTLER, G. D., and MERRIAM, J. C., *Boston Med. and Surg. Jour.*, 1925, xciii, 550.
 EVANS, W. A., *Brit. Jour. Surg.*, 1929, xvii, 34.
 MILLIGAN, E. T. C., and SIMON, G., *Brit. Med. Jour.*, 1931, i, 1114.

A CASE OF VESICAL PAPILLOMA WITH WIDESPREAD METASTASES.

By M. F. NICHOLLS,

ASSISTANT SURGEON TO ST. GEORGE'S HOSPITAL, LONDON.

It is often difficult to decide whether a vesical papilloma should be regarded as benign or malignant. All stages from completely innocent papilloma to rapidly infiltrating papilliferous carcinoma are seen. A proportion of benign tumours (25 per cent according to Ewing¹), or their local extensions or recurrences, may sooner or later become malignant.

The malignant villous tumour, however, does not usually spread beyond the bladder before it has killed the patient. If extension does happen, it is nearly always by direct spread to the pelvic tissues, or by the lymphatics to the pre-aortic glands. Distant metastases are rare, and all writers agree that the widespread skeletal deposits, so common in prostatic carcinoma, are almost unknown in vesical carcinoma. With growths that are both macroscopically and microscopically benign extension is confined to deposits within the bladder, unless an incautious operation allows implantation in the abdominal wound.

In the following case the very unusual behaviour of an apparently benign vesical papilloma seems worthy of report.

CASE REPORT.

W. C., age 43, was admitted into St. George's Hospital in April, 1926, having had hæmaturia on and off for a year. Cystoscopy showed extensive papillomata, but hæmorrhage was too profuse for diathermy. Accordingly the bladder was opened suprapubically, one large papilloma was excised, and several smaller ones destroyed with the electric cautery. The patient made a good recovery. After discharge from the hospital he failed to report for cystoscopy in spite of several recurrences of hæmaturia starting from August, 1927. Eventually, in July, 1929, he reappeared on account of pain and swelling of the right knee, noticed for two months, and was admitted.

ON EXAMINATION.—There was effusion into the right knee-joint, and tenderness over the internal condyle of the right femur. Movements of the joint were painful and limited. There was a scar in the thigh and an irregularity of the lower third of the femur due to compound fracture from a gunshot wound during the Great War. The Wassermann reaction was negative. X-ray photographs revealed no active lesion of the bones, but an old fracture of the distal third of the femur, united with overlapping and backward angulation of the fragments. Cystoscopy showed a very large villous tumour covering the posterior wall of the bladder.

OPERATION.—On Sept. 9 a papilloma the size of a clenched fist was excised by suprapubic cystotomy.

SUBSEQUENT COURSE.—Healing and recovery of bladder function was uneventful, but the patient remained ill. The right knee became progressively more swollen and painful. X-ray photographs taken on Sept. 19 showed destruction and decalcification of both femur and tibia, with deposition of new periosteal bone. The appearances suggested neoplasm.

On Sept. 24 arthrotomy was performed for diagnosis, and a small piece of tissue removed. The patient's condition deteriorated very rapidly, the tumour of the knee increased, and on Oct. 2 a mass was felt in the right iliac fossa. This also grew very rapidly, even in the short time before his death on Oct. 10, 1929.

POST-MORTEM FINDINGS.—

General Condition.—Wasted. There was a large bony lump on the inner side of the right knee-joint with a healed incision over it. There was a discharging sinus in the suprapubic scar.

Pleura, etc.—A few adhesions were present on the left side. On the sixth right rib near its vertebral attachment there was a lump the size of a pigeon's egg.

Lungs.—The right lung was normal. In the apex of the left lower lobe there was a node of growth the size of an orange.

Heart and Pericardium.—The pericardium was normal. The heart was small and atrophic, showing brown atrophy.

Abdomen.—No peritoneal exudate. A large swelling in the right iliac fossa stretched the iliacus and psoas muscles into a thin sheet over it. The ureter and the anterior crural nerve were also stretched on its surface. The mass, which was apparently arising from the ilium, was about the size of a coconut, and, on section, contained a quantity of old and new blood-clot, and had a framework of bony spicules. It extended into the gluteal region through the crest and blade of the ilium, which were completely destroyed.

Liver, Spleen, and Pancreas.—These organs were healthy.

Kidneys.—Both were congested. The right contained some small yellow calculi, but was otherwise healthy.

Bladder.—Contained thick yellow pus. The wall was inflamed and congested, but there was no sign of neoplasm.

Alimentary Tract.—Healthy.

Right Femur.—There was an old united fracture of the lower third. On the outer side of the bone at the junction of the upper and middle thirds, and again at the junction of the lower and middle thirds were two similar

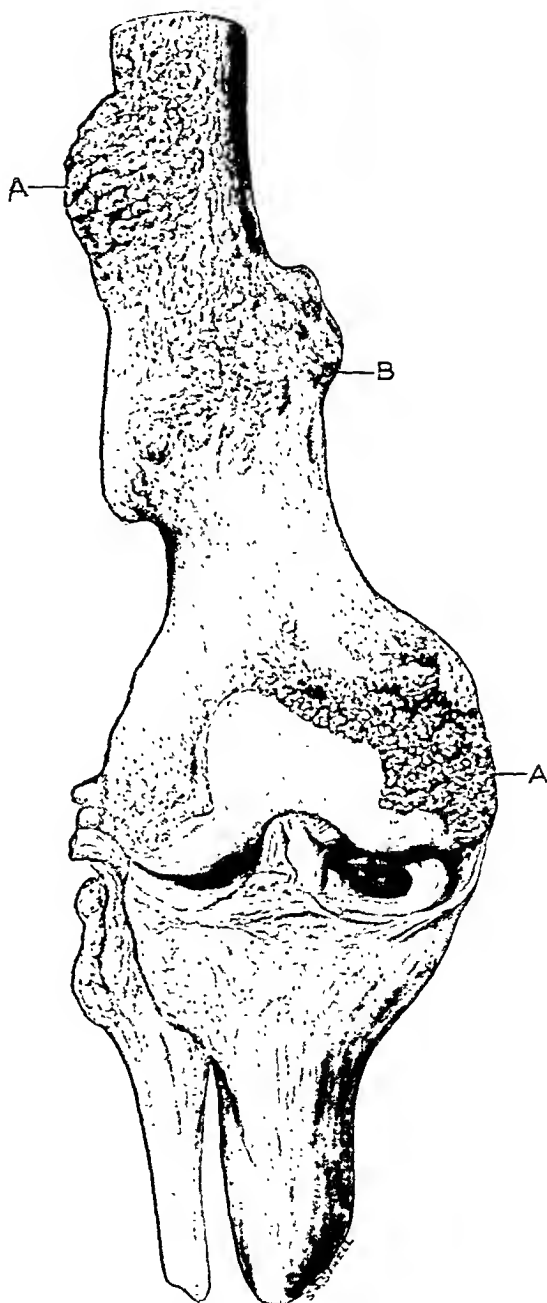


FIG. 72.—The bones of the right knee-joint, showing deposits of growth (A) and an old united fracture (B) ($\times \frac{1}{2}$)

oval masses, about the size and shape of half a hen's egg. Their surfaces were roughened, and their consistency varied, being bony hard and soft in different parts.



FIG. 73.—The right hip and pelvis, shewing mass in iliac fossa (A), and deposit in femur (B). ($\times 1$.)

There was no lesion of the knee-joint itself, but there was a mass of fleshy growth involving the lower end of the femur and the upper end of the tibia lying beneath the internal lateral ligament. (*Figs. 72, 73.*)

VESICAL PAPILLOMA WITH METASTASES 111

MICROSCOPICAL FINDINGS.—

1. Sections of the tumour removed in 1926 showed (according to the report made at the time) a typical vesical papilloma with no evidence of malignancy. These sections are unfortunately not now available.

2. Sections of the tumour removed from the bladder on Sept. 9, 1929, show a transitional-cell papilloma. The arrangement is a little irregular, but there is no sign of invasion of the deeper layers, and the individual cells are of uniform shape and size, and show no mitosis. (*Fig. 74.*)

3. Sections of the tissue removed from the right knee on Sept. 24, 1929, show



FIG. 74.—Photograph of a section of the tumour removed from the bladder in September, 1929, to show the general innocent arrangement. Note the absence of infiltration of muscular coat. ($\times 3$).



FIG. 75.—Microphotograph of section of tumour from the ilium showing papillary processes in a bony framework. ($\times 50$).

a transitional-cell papilloma exactly similar to the one from the bladder.

4. Sections of tissue removed post mortem :—

a. Bladder.—Sections of the bladder wall in the region of the removed tumour show no evidence of neoplasm whatever.

b. Lung, Rib, Pelvis, Femur, and Tibia.—Sections of the tumours of these organs all show a similar microscopical picture, that of a transitional-cell papilloma. Regular papillary processes form the bulk of the tumour in each case, the individual cells are regular in shape and size, and mitosis is absent. Ossification is evident in the sections from the bony tumours. (*Fig. 75.*)

DISCUSSION.

The following points suggest that the tumour in this case was what is usually regarded as the innocent type of vesical papilloma :—

1. Macroscopic appearance.

2. Multiplicity. It is generally held that tumours which are early multiple do not invade the bladder wall, although they may recur or extend on its lining with great rapidity.

3. Reaction to treatment. If a malignant papilloma is inadequately treated, recurrence is usually rapid and obviously carcinomatous. Yet, after local excision and cauterization in 1926 there was an interval of fifteen months

before any symptoms recurred, and the tumour excised in 1929 was single and non-infiltrating. A month after this, at post-mortem, no neoplasm could be found in the bladder wall.

4. Histology. It is notoriously difficult to be certain of the innocence of vesical papillomas by microscopy, but the appearances were entirely benign on both occasions.

On the other hand, wide dissemination by the blood-stream is so rare a complication of vesical carcinoma that it alone can hardly be held to denote a change in the character of the tumour. According to Colston,² metastasis to bones does rarely happen in bladder cancer, the bones of the pelvis and lower spine being early extensively involved. Operation or other therapeutic agencies (such as radium) may result in extensive and widespread metastases from a tumour which has shown no prior evidence of implantation. This, he holds, illustrates the fact, long recognized, that bits of benign papilloma, transplanted, always become malignant and infiltrate extensively. In the case under discussion, although the growth of the deposits was remarkably rapid and destructive, their microscopic picture, like that of the parent tumour, was not one of carcinoma.

It is certain anyway that cells gained access to the blood-stream, and it is of interest to consider when and how they did so. There are objections to the theory that they were introduced at either of the suprapubic operations. If at the first, it has to be explained why the growth of the emboli should have become suddenly and fatally accelerated after quiescence for three years. It is more reasonable to blame the second cystotomy for the events which so rapidly followed it, but some explanation must then be found for the pain and swelling of the knee which preceded this operation by two months, and for which no cause other than neoplasm was discovered either ante or post mortem. On the whole we must conclude that tumour cells entered the circulation independently of operation, from the recurrent growth some months before the patient was admitted to the Hospital in July, 1929.

It is difficult to understand how this could happen in a papilloma unless some few cells at least had lost their innocent nature. Possibly a fairly large vein of the tumour pedicle was eroded and gave them entry. The whole picture here resembles some cases of multiple thyroid metastases, and, as pointed out by Dunhill,³ malignant adenoma of the thyroid may erode blood-vessels and give rise to distant metastases early in its career, when it is small and confined to its capsule, and thus clinically benign.

According to Young,⁴ villous growths of the bladder have produced metastases by the blood-stream when apparently benign, but I have been unable to find the record of a comparable case.

I wish to thank Mr. C. H. S. Frankau and Mr. G. R. E. Colquhoun, under whose care this patient was, for permission to record the case.

REFERENCES.

- ¹ EWING, *Neoplastic Diseases*, 1928, 3rd ed., 910.
- ² COLSTON, *Nelson's Loose-leaf Living Surgery*, vi, 401.
- ³ DUNHILL, *Brit. Jour. Surg.*, 1931, July, 83.
- ⁴ YOUNG, *Practice of Urology*, 1926, i, 563.

THE SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM.

(The Bradshaw Lecture, delivered before the Royal College of Surgeons of England, December, 1932.).

By G. E. GASK,

SURGICAL UNIT, ST. BARTHOLOMEW'S HOSPITAL.

FIFTY years ago, on Dec. 13, 1882, Sir James Paget delivered the first Bradshaw Lecture in this theatre. Before beginning his lecture he paid a graceful tribute to William Wood Bradshaw, a Fellow of this College, in whose memory the lecture was founded by his widow. Paget took as the title of his paper, "Some New and Rare Diseases". I do not wish to comment on the subject-matter of the paper, but it is an appropriate time to recall some of his remarks, which seem almost prophetic, concerning our Museum and College.

You will remember that Paget gave devoted service to our Museum and that he believed whole-heartedly in its value as a source of study and a stimulus to research. This is what he said fifty years ago¹: "I feel that the collection is one which all we members of the College may feel personal pride in calling our own, and should feel a personal duty to enrich. And its utility is being constantly more appreciated. I have often been made happy by the contrast which I have seen while working at the new edition of the catalogue. While I was writing the last edition, between thirty and forty years ago, scarcely a student ever entered the museum. Hour after hour I sat alone. I seemed to be working for no one but myself, or for nothing but the general propriety that a museum ought to have a catalogue, though no one might ever care to study from it. Now, and for some years past, a day rarely passes without many pupils and others being at work in every part of the museum.

"All this is good, but much more is to be done. Our museum should be, even more than it is, the centre in which all pathologists may find help in searches after that which is not yet known. For many years, even from the beginning, the anatomical and physiological departments of our museum have been not only a noble collection of specimens, but through the renown and learning of its conservator, a great centre of teaching."

Paget evidently had firmly in his mind the idea that this College should be more than a storehouse of dry bones and a vast collection of bottled specimens. He believed that it should be an active centre for the advancement of surgical knowledge and an inspiration to all those who seek "after that which is not yet known". This dream of Paget's seems to be coming true. The workrooms are now full. A number of promising young men are busily engaged in various problems under the direction of our beloved Conservator. Through the influence of our late President, Lord Moyrihan,

the College has been enriched by a number of research scholarships. And lastly, through the great-hearted and far-sighted action of one of our senior Fellows, we have become the owners of the Buckston Browne Research Farm, with its laboratories for Surgical and Biological Research and Education.

Paget surely would have been pleased with these great developments, but he was not the only one who had the idea. The scheme originated, we may believe, in the fertile brain of John Hunter, the creator of the Museum. Hunter did not collect his specimens out of idle curiosity. The collection was not the real object of his work, the museum was the storehouse or reference library, while the workroom was his study. As Trotter said in his Hunterian Oration, the greatness of Hunter lay in his foundation of a school, and he laid the foundation perhaps better and more truly than he knew, for his influence is still felt and is as active to-day as it ever was.

The fifty years which have elapsed since Paget gave the first Bradshaw Lecture have witnessed the greatest advances in surgery the world has ever known. Discovery has followed discovery, and progress has been so rapid that we may be justified in saying that we are living now in the Golden Age of Medicine. In surgery these advances have been made possible by the work of Pasteur, amplified by Lister, himself a Bradshaw Lecturer in 1887, who, as Lord Moynihan once said, made operations safe for humanity.

If one analyses the advances that have been made, it is evident that following the introduction of asepsis, they have been largely due to developments of instruments and of methods of examination, and by these means to improvements of technique, so that little by little every region of the body has become accessible to surgery. This is a part of the truth only, for the development of a safe technique has enabled surgeons to gather much valuable information concerning the early stages of pathological processes, whereas previously they had to rely on the end-results as revealed in the autopsy chamber.

So much for the past. What of the present and of the future? If one reads the portents correctly, there is evidence that a change is coming over the art of surgery. It is becoming less of a craft and more of a science. The minds of surgeons are showing a leaning towards physiology, and surgery is becoming less extirpatory, more conservative and preventive. In other words, the operating-theatre is no longer only a place for the safe removal of diseased organs; it is becoming a scientific laboratory.

This young growth of preventive surgery should be fostered, and it is for this reason that I am taking as the subject of this lecture the surgery of the sympathetic nervous system. It is a vastly intriguing study, for not only is it opening up a new field for surgery, but it is also playing an important part in unravelling the mystery of the anatomy and physiology of this intricate system.

This subject, though still young, has already accumulated round it a mass of literature, and you may expect me to give you a conspectus of all the work that has been done and the history of the workers. It is quite impossible to attempt it in the scope of this lecture, and the reader is thereby spared an impressive bibliography. The endeavour of this lecture is to set out a straightforward narrative of the work that my colleagues and I of the

Surgical Unit of St. Bartholomew's Hospital have done, to tell you our experiences, and to show you some of the results. To attempt a record of my indebtedness to other writers would take too long; let it suffice to say that my creditors are too many to mention and the liability beyond my discharge. Yet I must take this opportunity of expressing how much I owe to the staff of the Surgical Unit and to the Departments of Medicine, Anatomy, and Physiology, without whose help this work could not have been done.

PERI-ARTERIAL SYMPATHECTOMY.

Our first adventure into this new field of surgery was an operation known as peri-arterial sympathectomy. We were incited to this by the example of Leriche, who was made an Honorary Fellow of this College in 1927. Leriche assumed the mantle of his master, Jaboulay, to whom much credit for initiating the work must be given. It is to be regretted that his promising life was brought to a premature close as the result of a railway accident. I cannot help referring also to one Englishman—Alexander, of Liverpool, probably the pioneer of all surgery of the sympathetic nervous system. His work received scant attention at the time, but it is bearing fruit now.

The first peri-arterial sympathectomy done by us was in 1924 on a patient with one blue foot which was associated with great pain. This man had a three years' history of progressive and disabling pain in the right foot. While he was lying in bed both legs appeared normal, except that in the right leg neither the posterior tibial nor the dorsalis pedis artery could be felt pulsating, while those in the left foot could. On standing or walking the right foot and ankle became intensely blue and painful, but on lying down they quickly resumed their normal colour. The condition was then diagnosed as 'endarteritis obliterans hebraica', though in the light of our present knowledge this must be regarded as incorrect. It might be more correctly named 'acro-cyanosis', which again is only a name given to a condition of the pathology of which we are ignorant.

One inch of the adventitia was stripped off the right femoral artery in Scarpa's triangle. Immediately after the operation the right leg became palpably warmer, and the patient was cured of his complaint, was able to return to his work, and has not had any return of his trouble up to the present time (1932).

Unfortunately we have never been able to state with any accuracy what disease the patient was suffering from. It looked as if he suffered from a spasmodic constriction of the arterial supply of the foot, and that this constriction was released by division of the vasoconstrictor sympathetic nerves, which at that time we presumed (though we know now erroneously) to be running down the limb in the outer coat of the femoral artery.

Encouraged by this promising start, a series of patients were treated by peri-arterial sympathectomy, or by stripping the main artery of its adventitia (a more correct description of the operation). The patients thus treated were afflicted, some with incipient gangrene due to arterial degeneration with occlusion of the lumen, some with endarteritis obliterans, and others with

painful stumps (causalgia). In none of these patients, excepting those with painful stumps, was any result achieved at all comparable to the first case described above. A description of these patients has already been published.²

This form of operation was then discontinued, because we came to the conclusion that it was based on a wrong premise. We had thought that the vasoconstrictor fibres supplying the vessels of the lower limbs came from the abdominal plexus and ran down the vessels to their termination, and that stripping their coats divided these fibres which are invisible to the naked eye. Gaskell and Langley many years ago proved that in the cat the vasoconstrictor nerves are carried to the blood-vessels via the peripheral systemic nerve trunks. Now Woollard³ showed, conclusively in our opinion, that in man also the vasoconstrictor fibres reach the arteries of the extremities by the peripheral motor and sensory nerves and not as we had imagined originally, and that we must look for some other means to effect our purpose.

Though this operation of stripping the systemic vessels of their outer coat (peri-arterial sympathectomy) has fallen into disrepute and mostly been abandoned, it is worth while remembering that from its use some small pieces of information have been gleaned, and while at the moment we do not fully understand their import they may later furnish a clue in the final *dénouement* of an intricate puzzle. Sir Thomas Lewis, when shown these patients, said, "You have certainly done something, though I do not know what." We think, therefore, that they are worth recording.

The points which were noticed in almost all the patients treated by peri-arterial sympathectomy were these:—

Firstly, the operation was followed by a rise of temperature of a degree or two in the affected limb. This rise was temporary only, disappearing after a few days or a few weeks. It was supposed at first that this rise of temperature was due to division of the vasoconstrictor fibres, which allowed dilatation of the peripheral vessel and consequent increased arterial flow. As stated above, this view must now be held to be erroneous and another explanation sought. We do not offer any; we merely record the fact. The second point we noticed was that the operation was followed by a rise in the leucocyte count in the blood from the affected limb as compared with the opposite limb. Many criticisms may be levelled at this observation, but it has been noticed by others, notably by Leriche. The third point noticed, and one which may still justify the continued use of the operation, was relief of pain. This is an important point, but very difficult to explain on physiological grounds. We know that the blood-vessels are sensitive and that painful sensations originating in arteries reach the brain, but we have no knowledge of the path by which they travel. Because a peri-arterial sympathectomy has in some instances been followed by relief of pain, it has been suggested that afferent sympathetic fibres run up the sheath of the big vessels to the abdominal plexus and thence through the sympathetic trunk to the spinal cord and so to the brain. This is mere supposition and is not based on anatomical or physiological evidence.

One further point of histological and perhaps of clinical interest may also be noticed. In some cases of long-standing vascular disease it was observed that gross changes were sometimes met with in the external coat

of the vessels. These changes consisted in great thickening and vascularization of the adventitia, so much so that this coat appeared to the naked eye as flecked with red patches. On microscopical examination it was seen that the external coat was permeated with small vessels, and there were present also numbers of giant cells, giving the impression that the external coat had been the subject of long-standing inflammation.

It now became evident, in view of our better knowledge of the anatomy of the nerve-supply of the peripheral vessels, that if we desired to secure the sympathetic denervation of a limb, the plan of operation had to be altered. To secure complete sympathetic denervation of the vessels of an extremity it is necessary either to cut all the grey rami through which the sympathetic fibres reach the peripheral nerves which send branches to the blood-vessels, or else to extirpate the corresponding part of the sympathetic trunk.

Anatomy.—May I break off from my story a moment to refresh our minds as to the general make-up of the sympathetic nervous system and to show how the vasoconstrictor fibres reach the blood-vessels?

The sympathetic nerves, which are linked on to the cerebrospinal nervous system, are distributed to the internal viscera, to the heart, the blood-vessels, and to the unstriped muscles of the body. For convenience this great sympathetic system, which has innumerable links with the cerebrospinal nerves, is divided into two main groups: (1) The two principal ganglionated cords; (2) The great prevertebral plexuses from which issue the efferent sympathetic fibres which are distributed to the viscera.

Though one talks of the sympathetic system as having an entity of its own, it is very important to remember that it is a part only of the nervous mechanism, and, as Bayliss says, "The sympathetic system is not an independent system, but an outflow of efferent fibres from particular regions of the central nervous system"; it is supplementary to and co-ordinated with the central nervous system.

It is interesting to speculate on the origin of the sympathetic system and to compare it with the nervous system of the lower animals. One cannot do more here than mention that in the more primitive animals, such as the worms and the cockroach, the nervous system lies on the ventral surface of the body, and not, as in vertebrates, on the dorsal side. In which period of the development of animal life the change over from a ventral nervous system to the dorsal system of the higher animals occurred is not known. It has been suggested that the sympathetic system of man represents the primitive nervous system of lowly animals, and that our dorsal nervous system of vertebrates has been grafted on to it. I am not qualified to express an opinion. All I can say is that modern anatomists tell me that there is no foundation for this suggestion.

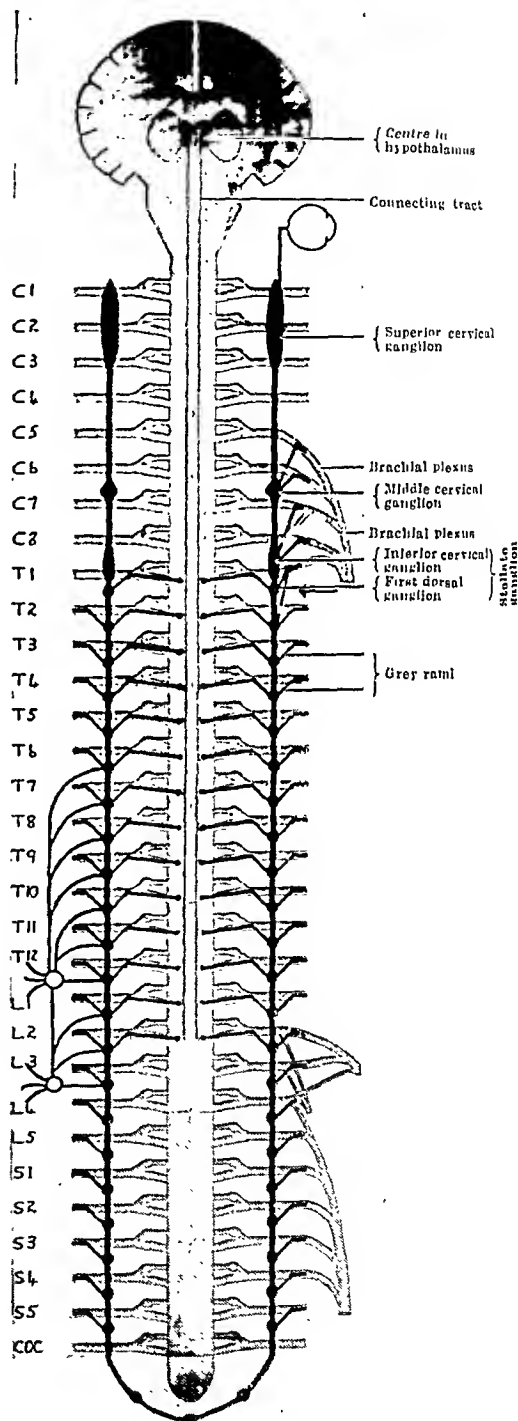
The foundation of the sympathetic system lies in a number of small medullated nerves which take origin from cells in the lateral horns of the spinal cord, and travel through the anterior roots of the spinal nerves and enter the sympathetic system by slender white communicating rami, to terminate in synaptic relation with the nerve-cells found in the sympathetic ganglia. These communicating cords are the white rami communicantes, or, as they are more commonly called, the preganglionic fibres. These cells are

now believed to be governed by centres in the base of the brain, in the lateral horn of the spinal cord, close to the hypothalamus, and it is thought that a tract of fibres connects these centres with the ganglia in the lateral horns. Demonstrations in substantiation of this point were given this year in this College by Sir Charles Ballance and Professor Beattie.

The proper sympathetic or post-ganglionic fibres, mostly non-medullated, arise from the cells in the sympathetic ganglia. These fibres run in two ways: (1) From the ganglia to the cerebrospinal nerves, these communicating cords being called the grey rami; (2) To the various plexuses, whence they are distributed to the viscera.

FIG. 76.—Diagram to show the sympathetic outflow in man.

The great ganglionated cords are two in number and consist of a series of ganglia with short intervening chains. These ganglionated cords extend from the base of the skull to the coccyx. The ganglia are nearly equal in number to the vertebræ except in the cervical region, where there are only three. The ganglia are severally connected with the anterior divisions of the spinal nerves by: (1) The white rami, which are composed of medullated fibres flowing out of the spinal nerves into the sympathetic ganglia—that is to say, preganglionic fibres; (2) The grey rami, composed of non-medullated fibres which are the sympathetic fibres passing from the sympathetic ganglia into the cerebrospinal nerves, and are called postganglionic. It is in these fibres that we are interested at the moment, and in them are



contained not only the vasoconstrictor fibres, but also sympathetic fibres to the sweat glands, hair muscles, and the eye.

As shown by Gaskell, white rami communicantes are not furnished by all the spinal nerves. They are given off by the spinal nerves from the second dorsal to the second lumbar nerves inclusive. There is no direct outflow of sympathetic impulses from the spinal cord into the first dorsal nerve, the eight cervical nerves, the lower three lumbar nerves, and the sacral nerves. Consequently the three cervical sympathetic ganglia must receive their branches from thoracic segments of the spinal cord.

Though there are no white rami to the cervical and first dorsal nerves, they get their sympathetic fibres, but not in a segmental way. The majority of the preganglionic fibres turn either up or down in the sympathetic chain, and run for varying distances within it before ending in the ganglia. The cervical sympathetic trunk is composed entirely of sympathetic fibres, coming from the spinal cord, through the white rami of the upper thoracic spinal nerves, and ascending to terminate in the cervical sympathetic ganglia.

These are important facts to remember when considering possible operations for the relief of conditions primarily due to vasoconstriction, such as Raynaud's disease.

The tracts of the sympathetic fibres within the spinal cord and their connection with the base of the brain, and the central mechanisms concerned in the control of nerve impulses through the preganglionic sympathetic neurones, do not fall within the scope of this paper. They are indicated in the diagram (*Fig. 76*). Their anatomy and physiology are the subject of hard work at the present time by various observers, and we may hope to hear more of this before long.

After this digression into the anatomy of the sympathetic nervous system, I will now return to the story.

We have reached the point where we had realized that peri-arterial sympathetomy, though it accomplished something, did not yield the desired results, and further that anatomists and physiologists had convinced us that in order to effect a sympathetic denervation of peripheral arteries it would be necessary to cut off the sympathetic supply at its source. That is to say, it would be necessary to divide the grey rami, through which the sympathetic fibres reach the nerves sending branches to the vessels, or else to extirpate the corresponding portion of the sympathetic trunk.

The work of Jonnesco, of Bruning, of Hunter and Royle, of Adson and Brown, and others, had stimulated interest in this form of surgery. We, in our hospital, under the guidance and stimulus of Professor Woollard, were now induced to abandon the pursuit of the operation of peri-arterial sympathetomy and to seek to denervate the peripheral blood-vessels by dividing the sympathetic fibres in the neck.

In the first instance, Professor Woollard set us the task of dividing the grey rami communicantes passing from the cervical sympathetic ganglia or cord to the fifth, sixth, seventh, and eighth cervical and the first dorsal nerves—that is to say, the cords of the brachial plexus were to be deprived of their sympathetic supply.

Case 1 (A. A.).—The subject was a lady, aged 32, who presented the typical appearances of Raynaud's disease. She had been healthy until the age of 16, when her fingers and toes began to go numb in cold weather, and she began to get whitlows round the tips of the fingers. This condition became progressively worse, and whenever the hands were exposed to cold they became white or mottled blue and white. Some experiments were made by Mr. J. Hunt to see if the 'attacks' of 'cyanosis' could be modified by injecting the nerves of the hand with novocain. Accordingly 3 c.c. of 2½ per cent novocain were injected into the tissues round the left median nerve 2 in. proximal to the distal end of the radius. In half an hour anæsthesia had reached its maximum. An 'attack' was precipitated by exposure of the hand to cold, with the result that the portion of the skin of the hand which was anæsthetized remained warm and red, while the non-anæsthetized area became dark blue. A few days later a control experiment was done on the same hand, only this time sterile water was injected round the median nerve instead of novocain. An 'attack' was again precipitated by plunging the hand in water at 55° F., and in a few minutes the whole hand became blue. These experiments seemed to indicate that the vasoconstrictor nerves run in the systemic peripheral nerves, and that if they are paralysed by novocain, the constricting impulses which they carry are blocked. This experiment is again confirmation of the work of Woollard, that the sympathetic nerve-fibres reach the vessels of the limbs via the peripheral nerves, and do not accompany them throughout their length in their outer coat.

The operation for dividing the rami communicantes passing to the trunks of the brachial plexus was carried out through a 4 in. incision parallel to the clavicle and ½ in. above it. The scalenus anticus muscle was exposed and the anterior primary division of the 5th cervical nerve isolated, and one ramus communicans torn through with a blunt hook. Next the 6th cervical nerve was isolated and one ramus communicans divided. Three such branches connecting the 7th cervical nerve were divided, and two or three from the 8th cervical and 1st dorsal. Finally, the inferior cervical ganglion was exposed, and the dorsal sympathetic trunk was divided with seissors just below the communication with the 2nd dorsal nerve.

Immediately following this operation the right hand became warm and dry, whereas it had been cold and clammy; the palpebral fissure was smaller on the affected side, and the right pupil was smaller than the left. It was found also that if the two hands were immersed in cold water, the right hand remained pink, while the left hand became blue. In addition the blood-pressure and oscillogram readings were higher on the right than on the left side.

The patient was enormously pleased with the result, and begged that the opposite side might be treated; this was done at a later date.

Case 2 (R. T.).—The second case was that of a lady, aged 38, also the subject of Raynaud's disease, only differing from the first patient in the fact that the 'attacks' of 'cyanosis' could be precipitated either by cold or by emotion, and the 'attacks' caused by emotion could sometimes be cut short by taking a glass of port, or perhaps two, or a cup of hot milk.

An operation was planned on the same lines as in the previous case—namely, with the object of carrying out a cervical ramisection on the right side. The cords of the cervical plexus and the rami communicantes to the fifth, sixth, seventh, and eighth cervical and first dorsal nerves were avulsed, and the dorsal sympathetic trunk was severed between the first and second dorsal ganglia.

The result of this operation was not so good as the first. The right pupil became smaller than the left and there was slight enophthalmos, and the right hand became warmer and redder than the left. But the improvement did not persist, and a note taken five months later states that the condition of the right hand is not improved; the fingers of this hand go blue many times a day. The right hand is no whit better than the left. The enophthalmos of the right eye persists, and also the contracted pupil.

We learnt something from this case. The fact that the pupil became small and the palpebral fissure narrow showed without doubt that the sympathetic nerve-supply to the eye had been divided. The fact that the colour of the hand changed but did not persist—the fact that the operation did not fully but only partially achieve the end for which it was planned—indicates that the whole of the sympathetic supply for that upper limb had not been divided.

In order to discover the reason for this failure it is necessary to study again the anatomy of the part. On the accompanying diagram (*Fig. 76*) it will be seen that one communicating sympathetic ramus is depicted passing to each cord of the brachial plexus. In fact these rami are very small, difficult to find, variable, and often several instead of one as shown in the diagram. Therefore, if the surgeon relied on cutting these rami, it would not be unlikely that he would miss some. But we did more than this, for we divided the main sympathetic trunk below the stellate ganglion. We believed that we had divided the whole sympathetic supply to the arm, and yet the result seemed to indicate that this was not so, for vasoconstrictor impulses somehow were still coming through.

There is a reasonable anatomical explanation for this apparent anomaly of success in the first patient dealt with and failure in the second. Kuntz has shown that in 21 out of 48 cadavers a branch from the second thoracic spinal nerve sends a branch to join with the first dorsal nerve, and so it must be conceded that the second dorsal nerve forms a part of the brachial plexus in a fairly high percentage of cases. Sympathetic fibres are found in the branch from the second to the first dorsal nerve. Consequently it must be assumed that whenever this occurs sympathetic fibres may also be conveyed to the brachial plexus. The logical sequence of this argument is that, in order to make sure of securing complete sympathetic denervation of the upper limb it is necessary to divide the main sympathetic trunk *below the second thoracic ganglion*. This explanation also accounts for the fact borne out by clinical experience that extirpation of the inferior cervical or stellate ganglion does not always do away with the vasomotor nerves of the upper limb.

A further explanation for failure after division of the sympathetic trunk has been given—namely, that regeneration of the sympathetic occurs very quickly. This rapid regeneration is stated to occur in cats, and it may be true also for man, and it may account for a recrudescence of symptoms in Raynaud's disease after a period of improvement. This point will be referred to again when the results of treatment are reviewed.

Case 3 (A. A.).—The patient described in *Case 1* had now come in to have her left hand treated. She was so pleased with the result of the first operation that she begged to have the other side done.

This was considered to be a favourable opportunity to test clinically the correctness of the view that complete sympathetic denervation of the upper limb could be attained by excision of the dorsal sympathetic trunk from below the second dorsal ganglion up to and including the stellate ganglion and without touching the variable and elusive rami communicantes.

An operation was planned and carried out for the purpose. By an incision similar to that previously described the inferior cervical ganglion and the dorsal

sympathetic cord was exposed, and after the second dorsal ganglion had been identified, 1 cm. of the cord was excised above this ganglion.

The result fully justified the view of the anatomists, for the left hand became now warm and dry and the patient was much pleased with the result.

By these steps we were led to the belief that the best operation for the relief of Raynaud's disease was excision of a portion of the thoracic sympathetic trunk from below the second dorsal ganglion up to and including the stellate ganglion—the total length of tissue removed being rather more than an inch. This satisfies the requirements of the anatomists and physiologists, and we felt that removal of such a length of the trunk would not be likely to be followed by regeneration. Of this point we are not quite sure now, as the subsequent history of the patient will show later. At any rate this is the plan which we have adopted and used for all our subsequent patients.

I will now give a brief description of the operative technique and follow with a description of the cases.

TECHNIQUE AND RESULTS OF THORACIC TRUNK SYMPATHECTOMY.

We have adopted the anterior approach through the root of the neck. I am aware that this is opposed to the practice of many excellent surgeons, who prefer the posterior route and who expose the sympathetic trunk by excising a portion of rib. I do not proclaim the anterior route is the only one or the best one, but it is the way we have found successful. What may be

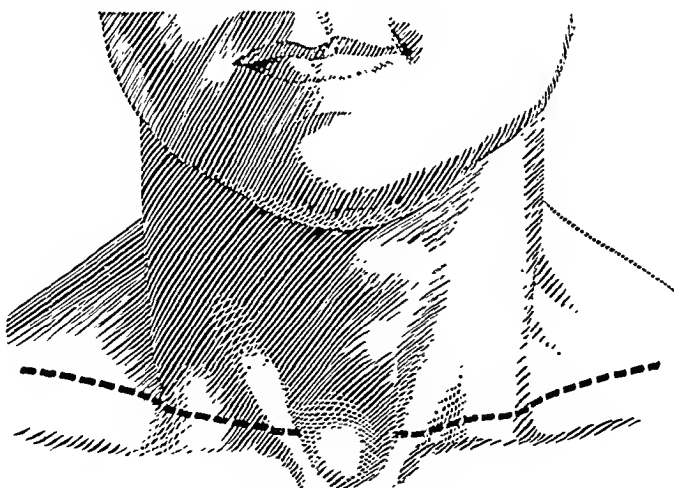


FIG. 77.

said of it is that it is a comparatively simple one, involving a delicate piece of anatomical dissection, though it is not to be recommended unless the surgeon has made himself familiar with the anatomy of the neck by a careful study of the cadaver. Personally I make it a practice to refresh my memory by a visit to the Anatomical Department before each operation. After a little

practice both sides can be done at the same sitting; there is no resection of ribs to be done and no important structure to be divided with the exception of the scalenus anticus muscle.

A 3-in. collar incision is made parallel to and $\frac{1}{2}$ in. above the clavicle (*Fig. 77*). This is deepened through the platysma until the omohyoid muscle is recognized. This muscle is of no importance and is divided and pushed aside. It is a landmark. The next landmark is the scalenus anticus muscle, which is cleared of its covering by a few strokes of the scalpel in the line of its fibres (*Fig. 78*). The phrenic nerve with its fascial covering, lying just anterior to the muscle, is

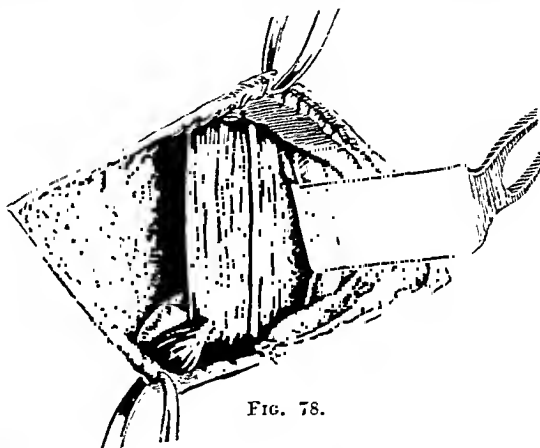


FIG. 78.

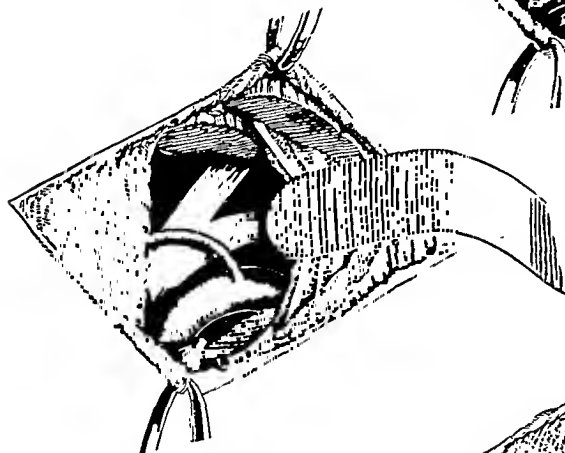


FIG. 79.

retracted towards the middle line. The scalenus anticus muscle is now divided transversely about $\frac{3}{4}$ in. above its insertion into the scalenus tubercle of the first rib

(*Fig. 79*). This is really the key to the operation which makes the subsequent part of the proceedings possible. A retractor is now placed so as to pull the subclavian artery out of harm's way downwards and towards the midline (*Fig. 80*). Then the forefinger of the left hand is pushed along the concavity of the first rib until the articulation of the head of the rib with the first dorsal vertebra is felt. With the forefinger of the right hand the dome of the pleura, together with the fascia covering it, is pushed downwards, and now the sides of the bodies of the first and second dorsal vertebrae are exposed. A

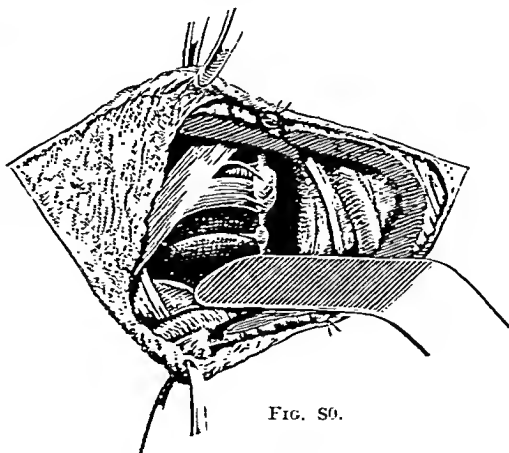


FIG. 80.

good light is needed here as the dissection is deep. The sympathetic trunk lies on the sides of these vertebræ, just medial to the articulations of the ribs with the vertebræ. With the tip of the left forefinger one feels the head of the first rib, and a little blunt dissection of the loose connective tissue lying on the side of the body of the vertebra soon reveals the trunk of the sympathetic, which can now be traced upwards and downwards.

We believe that the right thing to do is to divide the trunk below the second dorsal ganglion, for the reason, as has been stated above, that occasionally a branch is given off from the second dorsal sympathetic ganglion which communicates with the first dorsal spinal nerve. If this branch does exist, and if it is not divided, then some sympathetic impulses may escape from the spinal cord and the beneficial results of the operation may be diminished.

It may be asked, How does one know where to divide the sympathetic trunk? Can the ganglia be recognized with the naked eye? The answer is that it is often difficult to recognize the ganglia with the naked eye, even when the trunk has been dissected out of the body. There is little more than a slight thickening to indicate the position of the ganglia, but with the forefinger of the left hand it is easy to feel the head of the second rib and then with a pair of curved scissors to divide the trunk well below it. The trunk having been divided, it is easy to pull it upwards, to recognize the communicating branches, to expose the stellate ganglion, and to remove it.

The immediate effect of section of the sympathetic trunk is that the pupil becomes contracted, the eyeball sinks back, and the palpebral fissure is narrowed. This pupillary change is a useful test of the success of the operation. If it does not take place, one can safely infer that the sympathetic trunk has not been divided.

The operation conducted in this manner should not have any bad results. On one occasion I injured the pleura, causing a transient pneumothorax, and on another there was a temporary paralysis of the arm due to entirely unnecessary retraction of the brachial plexus. The operation is a pretty piece of dissection and can be done with the minimum of disturbance. In proof of this we can say that lately we operated on a lady of 78, the wife of a doctor and the mother of a doctor. Both sympathetic trunks were divided, the operation was completed in under one and a half hours, and the patient was healed and left hospital in a week's time.

It is time now to make a critical examination of the results of the operations which have been done. Seven patients suffering from Raynaud's disease have been treated in the manner described, four of whom have had bilateral sympathectomy done at one sitting.

The various tests which have been made before operation and the records of the physiological changes found after operation will not be described here. They are to form the subject of a Hunterian Lecture to be given at this College by Mr. Paterson Ross. I shall confine my attention to the results of the treatment on the course of the disease for which the operation was done.

These results may be divided conveniently into immediate and lasting. One can say without hesitation that the immediate results are dramatic and pleasing. Hands, which were previously blue, cold, and clammy, become in a few minutes pink, warm, and dry, and the attacks of cyanosis, often most

distressing to the patient, disappear. These pleasing alterations are accompanied by the characteristic eye changes—namely, contraction of the pupil, slight falling in of the eye, and drooping of the lid. This causes but slight inconvenience and as a rule is not complained of. Of far greater importance are the lasting results. Will the improvement be permanent, and will it hold through cold winters? That is the real test. As will be seen from the scrutiny of our patients, we are not able to guarantee a good permanent result in all cases.

Case 1 (A. A.).—The first patient has already been mentioned. This was a lady of 32, the subject of typical Raynaud's disease. The operation of ramiectomy was done on the right side in May, 1930. She was so pleased with the result that she came back in January, 1931, asking for the left side to be done. The thoracic sympathetic cord was excised on this side.

The immediate results on each side were eminently satisfactory. One is glad to say also that the good results have lasted through two winters. Sometimes the hands go a little blue, but they never become so bad as they were before. She is able to do her housework comfortably and has not had any attacks of cyanosis since the operation.

Case 2 (R. T.).—The second patient has also been mentioned as a partial failure from the start. Her condition was only slightly improved by the operation on the right side, and a few months later both arms were equally bad. She did not come back for a second operation.

We believe that this failure was due to the fact that we did an inefficient operation, by not dividing the sympathetic trunk sufficiently low and thus not securing complete sympathetic denervation of the arm.

Case 3 (E. M.).—The third case was that of a lady of 35, who at the age of 18 began to complain of blueness of the hands on exposure to cold. From the age of 22 the condition became steadily worse, and after the age of 29 changes appeared in the fingers, the nails becoming brittle and the finger tips stubby instead of pointed.

The case was regarded as one of typical Raynaud's disease, but there were in addition two features not present in the preceding cases. The first of these was a tightness and thinness of the skin about the nose and mouth suggesting scleroderma, and the second absorption of the bone of the terminal phalanges, leading to deformity of the tips. These additional features led us to regard the case as an advanced one.

The operation of thoracic sympathectomy was carried out on the right side in February, 1931, and she was so pleased with the result that she asked for the left side to be treated, and this was done in July, 1931. The immediate results were again satisfactory.

The last report, in November, 1932, is that the patient says that last winter the hands were rather better than they had been before the operation. This winter they are about the same, but they are distinctly blue and cold. The condition of the face is definitely improved, the tightness and thinness of the skin has disappeared, and the patient says her face feels warm instead of cold. On the whole she is glad she had the operation done. This may be regarded as a partial success.

Case 4 (M. C.).—The fourth case was that of a draper's assistant, aged 33, who began to suffer at the age of 25 and since then had been going from bad to worse. Both hands and feet were affected, though the hands were worse. Attacks of asphyxia and cyanosis were of daily occurrence even during the warm weather. The condition was so bad that she had to give up her work. This patient was regarded as an advanced case, and additional evidence to this effect was given by the fact that on the left side the radial pulse was barely palpable.

By this time we felt that sufficient experience had been gained to justify doing

both sides of the neck at one sitting. The same route was followed as has been described previously. On Oct. 20, 1931, the sympathetic trunk was divided below the second dorsal ganglion and a portion of the trunk excised up to and including the inferior cervical ganglion. In this instance I was unable with the naked eye to identify the second dorsal ganglion, and felt uncertain at first whether it had been removed. Examination of the part removed showed indubitably, however, that the cord had been severed where planned and that the second dorsal ganglion had been removed.

After operation the patient returned to the ward with both hands warm and pink, and the radial pulse, which previously could not be felt, was now easily palpable. She left the hospital in fourteen days' time well content with the result. In May, 1932, a bilateral lumbar sympathectomy was done to improve the condition of her feet, which were troubling her but had never been so bad as her hands.

The subsequent history of this patient is not good. It is sad to have to tell that the result has not been lasting. She was well during the summer of 1932 and was able to do her work as a draper's assistant, but with the onset of the cold weather in November, 1932, she relapsed; she has been compelled to give up her work as her hands are as bad as they were before. All one can say is that the feet, which prior to the lumbar sympathectomy were always cold, now give no trouble.

Case 5 (B. C.).—The fifth case was that of a married woman who said she had suffered since childhood from chilblains. Fourteen years ago she said she had a severe shock due to a bad post-partum hæmorrhage at the time of an air raid. Since that time she suffered from spasmodic attacks, in which the fingers, and to a lesser extent the toes, became first blue and then white and insensitive. She also complained of sores about the finger-tips which took a long time to heal.

Bilateral thoracic sympathectomy was done on her in November, 1931, the operation being the same as described in the last patient. This patient left hospital eight days after the operation, completely relieved of all her symptoms.

The last report, in November, 1932, is that the hands were perfect until the spring of 1932, when the fingers began to go blue again. Since the cold weather started last November the blueness and whiteness have increased, especially on the left side. The patient is able to do her work, and says she has nothing like as much pain in her hands as before the operation.

Case 6 (L. C. W.).—The next patient was diagnosed as suffering from Raynaud's disease, though admittedly it could not be described as a typical case. In the first place she was a lady of 78 who first began to complain of symptoms only nine months before her operation. Her chief complaints were that her hands were never warm, and that she had great pain in the arms and hands at night, burning sensations spreading from the elbow to the tips of the fingers. There was no evidence of any marked degree of arteriosclerosis and no other symptoms.

Owing to the difficulty of diagnosis I was reluctant to operate, and only did so at the urgent request of her husband, a very experienced physician, who was convinced that an attempt should be made to relieve the pain, which had become intolerable.

Bilateral thoracic sympathectomy was performed in June, 1932. The operation caused little disturbance. It was completed in one and a half hours, and the patient was able to leave hospital after eleven days.

The immediate result was good. The hands were warm, and while in hospital there were no further attacks of pain. At the end of five months after operation the patient's husband writes to me: "Emphatically she is improved as the result of the operation. She never has nocturnal pains nor any diurnal as before operation. There is never need to warm spoons and forks as was the case."

Case 7 (M. M.).—The seventh and last case of this series is a very interesting one. It is that of a lady of 34 who at the age of 22 began to suffer from 'chilblains', and in 1930 experienced attacks of blueness and numbness of the hands. In 1931 these attacks were more frequent, coming on in the summer as well as in the winter.

In September of 1932 a peri-arterial sympathectomy was done on the left brachial artery in a hospital in Scotland, without the least sign of improvement. When we saw her two months ago she presented the typical signs of Raynaud's disease. Attacks of cyanosis affected both hands, and the attacks could be precipitated by emotion or by immersion of the hands in cold water. There was no radial pulse on the left side, and the left hand was rather worse than the right.

Here was a chance to test the truth of the belief that the sympathetic vasoconstrictor fibres run to the peripheral vessels via the peripheral nerve-trunks and not in the adventitia of the big vessels. Accordingly a manœuvre was devised to demonstrate this point. An attack of cyanosis was precipitated by immersion of the hand in cold water, and while the hands were still blue the median nerve at the wrist was infiltrated with novocain. As the brachial artery had been stripped, if the vasoconstrictor nerves were taking the route of the artery then cocainization of the median nerve should have no effect on the vascularization of the hand. Whereas, if the vasoconstrictor nerves accompanied the peripheral nerves they would be paralysed, then the area of the hand supplied by the median nerve should become pink. The hand was watched with intense interest. In a few moments a faint change was observed, and then in five minutes the area supplied by the median nerve became a bright pink while the rest of the hand remained blue—a remarkable demonstration of the correctness of the statement made by Woollard and others. It was now possible to go a step still further and show that thoracic sympathectomy would secure sympathetic denervation of the limb. The operation was done on both sides and it was satisfactory to note that both hands became pink and warm.

It is too early, seeing that the operation was only done in October, 1932, to say what the late results have been; still, at the end of a month, although the weather has been cold, the good results persist.

ASSESSMENT OF THE RESULTS OF TREATMENT OF RAYNAUD'S DISEASE BY SYMPATHECTOMY.

It may appear presumptuous to attempt to draw deductions from seven cases. It is essential, however, that we should not be carried away by the glamour of a dramatic operation, and a careful analysis should be made of the actual results obtained.

The first point that emerges is that these operations have resulted in a better understanding of the anatomical distribution of the vasoconstrictor nerves. They have served as a confirmation of the work of the anatomists and physiologists. As regards the value of the method in the treatment of Raynaud's disease, it can be said that the immediate results are good. As regards the late results, the same cannot be said without qualification. Some are greatly improved, and others very slightly. The improvement is not of a lasting character in all cases.

The clinical results of sympathectomy in the patients having now been described and the facts as far as we know them having been stated, it is interesting to turn to speculation and wonder what is achieved when the sympathetic is divided. There is not the least evidence that the sympathetic nerves are themselves diseased, and it is clear also from examination of the patients that the disease, whatever it is, is not cured. It has been suggested that some unknown substance in the body, possibly endocrinal in origin, sends messages of a vasoconstrictor character along the conducting nerves and that severance of the nerves prevents the message being delivered. This seems too fanciful to be regarded seriously. It appears to us that the suggestion

made by Sir Thomas Lewis is the most helpful. He thinks that in Raynaud's disease there is some unknown condition which reduces the mean calibre of the small vessels, and that sympathectomy, by abolishing the vasoconstrictor impulses, results in a slight increase in the mean calibre and therefore a better blood-supply. This idea accords with the results of our operations. The future of the operation remains yet to be proved, but, if one may judge by the results obtained, it looks as if the vessels regain their power of constriction after a time and that unless the operation is done at an early stage it will have only limited use.

There is another point which requires elucidation. How do the vessels regain their power of constriction, as they seem to do? One suggestion is that the nerves regenerate quickly. In order to prevent this it might be well to divide the trunk with a cautery instead of with scissors, and this procedure we propose to adopt.

LUMBAR SYMPATHECTOMY.

Having had some success in the treatment of Raynaud's disease by division of the cervical sympathetic trunk, we turned our attention to the condition known as thrombo-angiitis obliterans. While the operation of peri-arterial sympathectomy was in fashion, we did a number of cases without registering any success, and so gave it up.

It does not seem reasonable to expect that a vessel which had already become thrombosed and obliterated could ever become patent and function again, and one would expect that in these conditions a lumbar sympathectomy would not prove useful. We thought, however, that, perhaps, if a lumbar sympathectomy could be done in the early stages of the disease, before occlusion of the vessels had taken place, the resulting dilatation of the arteries might avert gangrene of the limb and save amputation. So far as we have seen, this theory is not founded on fact.

The case we used as a test was that of an Englishman, aged 31, who had had his left leg amputated for typical thrombo-angiitis obliterans three months previous to admission. The patient came to us on account of a feeling in his right leg which reminded him of the feeling which preceded the gangrene in his left leg. The right leg showed but poor circulation, the skin was cold and of a blue tint. The dorsalis pedis and posterior tibial arteries could not be felt, but the popliteal was pulsating.

It is a comparatively simple procedure to denervate the lower limb, as will be seen from the diagram (*Fig. 76*). It is only necessary to excise the second, third, and fourth lumbar sympathetic ganglia with the intervening cord. This was done through a mid-line infra-umbilical, trans-peritoneal incision. After operation there was a temporary improvement, but then the patient became worse. Gangrene set in a few months later, the leg was amputated, and the man died.

It appears that in a disease like thrombo-angiitis obliterans the trouble is not stopped or even delayed by sympathectomy, and we shall have to look for some better method.

HYPOGASTRIC SYMPATHECTOMY FOR MEGALOCOLON.

We now come to the difficult and dangerous question of removal of the hypogastric sympathetic for disturbances of the colon. I say 'dangerous' deliberately, because there is a risk that if the operation is boomed it might be used indiscriminately and become a fashionable cure for constipation, and so bring the method into disrepute. The greatest care should be taken to record accurately which sympathetic fibres are divided at operation, and this is not always easy to do; and further, the subsequent history of the patients treated should be followed for a sufficient length of time to determine the true value of the treatment.

It will be seen by reference to the diagram that the sympathetic supply to the colon comes from the hypogastric plexus and that this can be removed without touching the lumbar ganglia. The nerve-fibres run in the loose connective tissue on the front of the aorta, and they spread along the course of the inferior mesenteric artery and also down the aorta to the two iliac vessels and into the connective tissue lying between the two iliac vessels in front of the vertebral column. One cannot identify all these branches with the naked eye, therefore the operator should aim to strip all the loose connective tissue off the front of the aorta from an inch above the inferior mesenteric artery, including an inch of that vessel, and then down the aorta to the two iliac arteries, and remove as well the loose connective tissue between them.

We have done the operation three times and the immediate results have been startling and aptly described by Lord Moynihan as too good to be true. I feel we must be very cautious before we accept them as true, for we are not sure as yet whether the results are lasting.

Case 1 (J. I.).—The first patient was a girl of 19, who suffered from constipation, retention of urine, with enormous distension of the colon. She could not remember ever having her bowels open without medicine, and as she grew older she would go days or even weeks without relief. As a child she would pass water only twice daily, and for three months she had to be catheterized, retention being complete. She was treated vigorously by the physicians for a long time and then referred to us as they could not keep her well. Physical examination revealed nothing beyond enormous distension of the colon.

The operation was performed in May, 1930. From the day of operation the patient began to pass her water at regular intervals without difficulty. Enemata and aperients were required until she began to get about again, after which time the bowels acted twice daily without assistance.

Eighteen months later she appeared healthy and of good complexion. Occasionally aperients were necessary, but with care the bowels could be kept regular. Barium enema examination showed that the colon was much less distensible than formerly. One may say without exaggeration that this patient has been greatly improved.

Case 2 (Nurse H.).—The second case was that of a hard-working hospital nurse, who had been through the Great War, and it was only in 1928 (following, in 1921, radium into uterus for menorrhagia; 1922, right oöphorectomy; 1927, subtotal hysterectomy and partial left oöphorectomy) that she noticed that she was becoming constipated and had abdominal distension. The bowels were only opened after large doses of medicines and enemata. She had practically to spend her life getting her bowels open. She had been admitted to Queen Mary's Hospital, Roehampton.

and was discharged as incurable. On the suggestion of Lord Moynihan she was sent to St. Bartholomew's Hospital for sympathectomy.

On admission she was found to be fairly well nourished, and had extreme difficulty in getting her bowels open; they could only be moved by enemata. In addition to that, she could go long periods without the slightest inclination to micturate; frequently she went twenty-four hours. Also, she had found that during the last two years her hands and feet got cold and blueish.

Operation (Nov. 6, 1931): Numerous old adhesions were found between the coil of small intestine and caecum, cervical stump, and the left ovary, which was cystic. All cellular tissue in the region of the bifurcation of the aorta and one inch of the common iliac artery were dissected away, and all the tissues between the two iliaes down to the shining membrane. The inferior mesenteric artery was identified and all the loose tissue stripped from it for an inch along its course.

The immediate results were most remarkable. The bowels were opened naturally on the second day after operation, and the patient micturated normally. She found herself ever so much better and expressed herself as delighted with the operation.

A year has now passed since the operation, and the patient is still pleased with the result. She is once again able to earn her living and to lead a normal life. With the help of simple laxatives she keeps herself fit.

Case 3 (H. P.).—This was a young girl of 14 who had had trouble in opening her bowels from the day of her birth: she was ten days old before her first action. Ever since she has had trouble, and sometimes six weeks passed without a motion.

At operation, on Nov. 15, 1932, the sigmoid flexure and ascending colon were enormously distended and thickened, assuming the appearance of a conger eel. The sympathetic supply of the colon was removed, as we believe, by the method described above.

So far all is going well, but it is too soon to say more.

As was said before, there is need for caution in assessing the results of these operations. They are still too few and too recent to justify our forming a good opinion. It is generally admitted that the immediate results are good, though why they should be good is a question we cannot answer. Is it the effect of the mind on the body, or does the removal of sympathetic stimuli to the unstriped muscle of the bowel render that organ resilient instead of spastic? Very little is known of the sympathetic supply to the bowel. We do not know if the sympathetic joins the plexuses of Auerbach and Meissner, and the part played by these great plexuses still wants explanation and offers a good field for research.

This brief survey of the surgery of the sympathetic system is admittedly inadequate. It is to be regarded as an adventure into the field of research, rather than as a conquest bearing tribute. As Paget said, it is meant to help in "searches after that which is not yet known", and if in making this contribution others may be incited to probe still deeper into the unknown, I believe that Mrs. Bradshaw, the founder of this lecture, would approve.

REFERENCES.

¹ PAGET, *Lancet*, 1882, ii, 1017.

² *Proc. Roy. Soc. Med.* 1927, xx.

³ WOOLLARD, H. H., "The Innervation of Blood-vessels", *Hcart*, 1926, xiii, No. 4.

PIED FORCÉ OR MARCH FOOT.

BY HAROLD DODD,

HON. SURGEON, KING GEORGE HOSPITAL, ILFORD; TO THE ROYAL HOSPITAL, RICHMOND;
AND ASSISTANT SURGEON, PRINCESS LOUISE HOSPITAL FOR CHILDREN, KENSINGTON.

"MANY obscure cases of injury to the foot without any history of traumatism in civil life will no doubt receive elucidation by the new facts brought to light by March Foot."²¹ Thirty-four years ago, this sentence concluded an editorial leading article on March Foot, yet the position to-day is not much altered.

The following case report and another from elsewhere²⁰ show unnecessarily radical treatment for this condition of Pied Forcé or March Foot, and probably even now, although orthopædic surgeons are familiar with it, it is not otherwise widely known and not usually described in the text-books; therefore this note may be justified, in the hope that similar mistakes may be avoided.

CASE REPORT.

Mrs. C. M., aged 51. No family. November, 1930. (Dr. Romanes' patient.)

HISTORY.—There was pain in the fore part of the right foot, centred at the shaft of the second metatarsal; it was aggravated by walking. The patient was in excellent health and had played golf daily until a month previously. She could not recollect any slip, strain, or accident during the several preceding years, either to her feet or other part of her body. Her past illnesses were trivial and not relevant.

ON EXAMINATION.—The patient looked healthy and fit, and weighed about 8½ stones. There was a diffuse patch of tender, painful, solid œdema over the shafts of the metatarsals, more prominent over the second, with a blushing of the skin and a little dilatation of the superficial veins. There was no tenderness on pressure over the spring ligament of the foot, nor of the attachments of the internal lateral ligaments of the knee-joint; no hallux rigidus or valgus; no metatarsalgia or gross callosities. No glands were palpable in the popliteal space or inguinal regions.

X-ray Examination.—Fig. 81 is a skiagram taken by Dr. Rhyndland Parry, who reported: "The distal end of the shaft of the second metatarsal shows periosteal reaction perpendicular to the shaft; there is some localized rarefaction. I cannot obtain any history of trauma. The appearances lend suspicion to a new growth (sarcoma)."

Owing to the element of doubt in this report a second opinion was obtained from an independent radiologist, Dr. Claude Goulesbrough, who reported: "This



FIG. 81.—Mrs. C. M. March Foot (? sarcoma) affecting the second metatarsal shaft. Note the short first metatarsal and the thickening of the shafts of the second and third metatarsals and of the basal phalanges of the third and fourth toes.

case is interesting and difficult; my opinion is that she has had an old united fracture and is now developing a periosteal sarcoma over the old callus which obscures the ordinary diagnostic appearances of new growth. I must admit that there is only a shred of evidence for this."

A month later Dr. Rhyndland Parry took another radiograph and repeated his opinion of a probable new growth. Two independent Wassermann reactions were returned negative, thus excluding a syphilitic lesion.

OPERATION.—In view of the suspicious radiological diagnosis of sarcoma, I advised a Lisfranc's amputation; this would eliminate the painful swelling and the possible new growth. I considered a more radical amputation would not materially help the prognosis, whilst local excision of the metatarsal, if it were the seat of a sarcomatous change, would be dangerous owing to the risk of growth implantation in the wound. Accordingly on Dec. 3, 1930, the classical Lisfranc's operation was performed and the patient made an uninterrupted recovery.

MACROSCOPIC SECTION.—The considerably thickened metatarsal consisted of dense greyish-pink bone throughout, with evidence of a slight irregular crack in the shaft, but none of localized growth. There was no softening; the whole bone was hard and sawn through with difficulty.

MICROSCOPIC SECTION.—Dr. Arthur Davies reported on the section (*Fig. 82*) as follows: "On the evidence afforded by the microscopic

FIG. 82.—Microscopic section of second metatarsal.
($\times 20$.)

sections examined, I am unable to find signs of a sarcoma. The lesion is, as far as my sections show, a rarefying osteitis with a chronic inflammatory periostitis."



FIG. 83.—Mrs. C. M.'s feet, showing fair appearance after right Lisfranc's amputation. These were the first pair of shoes, after two year's wear; an improved new pair, now being made, will be less noticeable.

POST-OPERATIVE PROGRESS.—The patient was fitted with boots, the front half of the right one being blocked out and fitted with a spring arch support, and she has recovered her walking well. Later she took to shoes similarly fitted, and has resumed her golfing activities during the past year, playing eighteen holes, four or five times a week, with her original handicap of 24 (*Fig. 83*). Her general health is good, and there is no sign of a recurrence either in the foot or radiologically in the chest.

I have come to the conclusion that this is a case of Pied Forcé on the following grounds: (1) The microscopical section; (2) The history of the affection (I think the patient probably developed her fracture through straining her feet at golf, which she played hard); (3) The after-history (it is two and a half years since the tumour was detected, and, had it been malignant, recurrence would now very likely have occurred).

This case is of further interest, showing that a Lisfranc's amputation, although nowadays seldom performed or advised, still has a useful place in surgery, the patient having a sound and presentable foot, able to withstand the strain of ordinary life, whilst being inexpensive and trouble-free to maintain.

THE LITERATURE.

In 1855 Breithaupt¹ described cases of persistently œdematous and painful feet occurring in soldiers, calling it 'Fussgeschwulst', i.e., traumatic inflammation of tendon-sheaths. In 1877 Pautat,² a French author, pointed out its occurrence in soldiers and that there was a marked periosteal proliferation; this latter observation was a clinical one, the help of X-rays not being then available. He further observed that later exostosis developed on the shafts of the second, third, and fourth metatarsals, that of the second being the commonest. In the same year Weisbach,³ in discussing the same condition, came to the conclusion that it was due to traumatic inflammation of the inter-metatarsal ligaments rather than of the tendons, calling it 'syndesmitis metatarsia'.

In 1888 Poulet⁴ concluded that the underlying cause was a rheumatic osteoperiostitis, and he did not agree with Pautat's theory that it was excited by the transverse crease of the uppers of heavy boots. In 1891 Martin,⁵ as a result of seeing 18 cases, decided it was due to synovitis and arthritis of the joints of the fore part of the foot.

In 1897 Busquet⁶ wrote a memoir upon ossifying osteoperiostitis of the metatarsals, and he divided them into three classes: (1) Direct primary immediate traumatic periostitis, due to repeated slight shocks in the fore part of the dorsum of the foot, largely owing to the transverse furrow in the upper leather of the shoe; (2) Indirect traumatic periostitis; (3) Diathetic periostitis.

Schulte,⁷ studying it this year (1897), recognized the fracture. In the same year Stechow,⁸ of the Prussian Guard, at Madrid discussed fracture of the metatarsals as a cause of swollen feet in soldiers. His observations are of considerable historic interest in that they were the first to be studied with the help of the X-rays. He had seen 36 cases. He pointed out that owing to the protrusion of the head of the second metatarsal beyond its

fellows, it consequently received the brunt of the strains exerted on the foot from the sides. Further that: (1) There was a fracture of the shaft of the metatarsals in nearly all cases; (2) The left was affected more frequently than the right. (3) Of 36 fractures, they occurred in the following order of frequency: second metatarsal 19, third metatarsal 14, fourth metatarsal 2, first metatarsal 1.

In February, 1899, Chapotot and Boisson⁹ further reviewed the condition. They found that: (1) Crepitation was always present; (2) The second metatarsal was always afflicted in their cases, the third occasionally suffering; (3) The fracture occurred at the junction of the middle and distal thirds; it was v-shaped, with the concavity forward, and never a transverse break; (4) Soldiers could still raise themselves on tip-toe, thus differentiating March Foot from arthritis. As an explanation for its occurrence the writers offered the greater fixity, length, and active function of the second metatarsal, these producing the condition necessary for a lever of the second order on a slender bone. They further said it might arise from arthritis and strains of the metatarsal articulations.

Thiele¹⁰ in 1899 studied 17 cases; he met them in three and a half months, all from one battalion.

The numbers of cases during these early years are surprising; the condition was very common amongst soldiers, perhaps owing to their severe and exhausting régime, which would certainly be heavier than in the present day.

Momburg¹¹ in 1904 believed that the prolonged elastic bending of the metatarsals resulted in an inflammatory reaction which was later followed by fracture. He showed that in 'uncomplaining feet' silent proliferations of the second and third metatarsals were present. *Fig. 84* shows a similar condition in a patient reporting with a fracture of the base of the first metatarsal.

In 1905 Kirschner¹² decided that all March Feet were due to fracture of the metatarsal shaft, and that the displacement was slight owing to the immobilization of the fragments by the surrounding structures. Further, he believed that the fractures occurred after the muscles supporting the metatarsals (long toe tendons and interossei) were exhausted, so that the strain fell undamped directly on the ligaments and bone. Later, Bähr¹³ in 1913 again studied the condition amongst soldiers.

In 1921 Deutschländer¹⁴ described 6 cases in civilians, all women, instead of the usual soldiers. Three of his patients ran a low febrile course, and he illustrated the slow callus formation—eight to nine instead of the usual three to four weeks. He considered that there was a low-grade hæmatogenous infection present, giving rise to a periostitis in the second and third



FIG. 84.—A weak but 'uncomplaining' foot showing: (a) A somewhat short but hypermobile first metatarsal, with posteriorly placed sesamoids; (b) Thickened second and third metatarsal shafts; (c) Fracture of the base of first metatarsal (the reason for the X-ray).

metatarsals. He remarked on the pain provoked by moving the second or third toes sideways, and pointed out that the site of fracture was that of the entry of the nutrient vessels of the metatarsal shaft (*Fig. 85*).

Sir Robert Jones¹⁵ in 1921 mentioned March Foot, and again with Lovett¹⁶ he refers to it as 'Pied Forcé' in 1923.

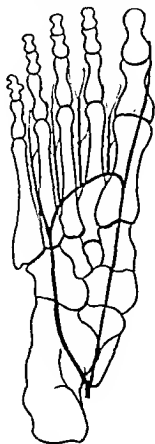


FIG. 85.—Showing the blood-supply of the shafts of the metatarsals. (After Deutschländer.)



FIG. 86.—The origins of the interossei muscles; note that the second, third, and fourth metatarsals are chiefly concerned. The dotted lines indicate the origins of the three plantar interossei, the continuous lines the origins of the four dorsal interossei.

In 1926 Jansen¹⁷ described 6 cases. He discussed the literature, and offered the explanation that the condition was due to anterior spastic flat-foot, i.e., spasm of the interossei muscles, resulting in œdema with lymph and venous stasis about the metatarsals, especially those where the interossei take origin, i.e., the second, third, and fourth (*Fig. 86*). This spasm of the interossei, denoted by pain and resistance on attempting to move the toes sideways on the head of the metatarsal, was also noted by Deutschländer, but without remark on its significance. Jansen said proliferation of the soft parts and periosteum follow with the laying down of new bone and increased density of the shafts of the second, third, and fourth metatarsals (*Figs. 81, 87*). (The case illustrated by Goldman¹⁹ shows this phenomenon, although he does not point it out; similarly, it is present in my case.)

Later, spindle-shaped formations develop on the metatarsal shafts, and these, Jansen explained by: (1) Callus around the fractures (*Figs. 81, 87*); (2) The organization of subperiosteal hæmorrhages, which readily occur owing to the congested periosteum being easily detached by a strong pull of



FIG. 87.—A 'weak' foot with a hypermobile first metatarsal, showing: (1) March Foot of the third metatarsal; (2) Thickening of the shafts of the basal phalanges of the second and third toes; (3) Thickening of the second and third metatarsals.

the interossei. He emphasized that these changes were located only to the site of the interossei attachments. With regard to this statement, I would point out the slight bulbous enlargements of the shafts of the first phalanges of the second, third, and fourth toes in the case of Mrs. C. (painful flat-foot, with thickening of the shaft of the second metatarsal, *Fig. 88*), also *Figs. 81, 87, 89*. The fluffy swellings on the basal phalanges are at the attachment of the fibro-aponeurotic canals of the long and short flexor tendons of the toes. "These fibrous canals are bounded above by the phalanges and below by fibrous bands which arch across the tendons and are attached on either side to the margins of the phalanges. Opposite the bodies of the proximal and second phalanges, the fibrous bands (vaginal ligaments) are strong and the fibres are transverse" (Gray¹⁸). This phalangeal thickening suggests unusual strain on these canals, probably from continued spasm of the flexor tendons.

Jansen explains the fracture as follows:—

1. Abnormal and excessive function of the interossei resulting in spasm, produces increased

hydrostatic pressure, especially lateral, around the shafts of the metatarsals. This is followed by "absorption of lime salts and thinning out of its constituent parts", with resulting 'brittleness'.

I would point out, however, that the X-rays show no sign of absorption or erosion of the shafts of the metatarsals; on the contrary, they are definitely denser and thickened, but with loss of definition of the lines of architecture or lines of stress (a weakening factor), especially of the second and third (the most frequently affected).

2. With the flattening of the transverse arch the second metatarsal, being longest, is pressed hardest on the ground in walking.

He assumes that there is a transverse arch of the fore part of the foot, but its existence is denied by Morton.

He admits that direct repeated trauma of the second metatarsal may excite the fracture. This trauma is considerable; it amounts to the weight of a man, say 140 lb., with a 90- or even a 110-lb. pack, in all over 2 cwt. This applied weight, plus the power of the 'push off' in walking, is borne by each foot sixty to seventy times a minute whilst marching (120 to 140



FIG. 88.—Mrs. C. A painful 'weak' foot showing: (1) Thickening of the shafts of the second and third metatarsals; (2) Fluffiness of the shafts of the basal phalanges of the third and fourth toes and of the second slightly; (3) Posteriorly placed sesamoids.



FIG. 89.—A 'weak' foot, showing: (1) Hypermobility short first metatarsal (space between first and second cuneiforms and posteriorly placed sesamoids); (2) A compensatory hypertrophy of the second metatarsal; (3) Thickening of the shafts of the second, third, and fourth basal phalanges; (4) Osteo-arthritis of the second metatarso-phalangeal joint.

paces per minute). I think that trauma must play the larger part in producing the fracture, and that the spasm of the interossei is a small contributory factor.

Further, with regard to the site of the fracture, Jansen's theory of spasm of the interossei, whilst not explaining its consistent location to the junction of the middle and distal thirds of the metatarsal, does offer a hint. The congestion around the metatarsals similarly affects their nutrient vessels, and thus causes the vascular foramina to enlarge, so establishing a constant weak spot. The nutrient vessels, according to Deutschländer¹⁴, enter at the distal two-thirds, this being the place where the fracture always occurs.

In 1928 Goldman¹⁹ reported the case of a pedlar aged 29 who developed Pied Forcé in his right foot. He carried an 80-lb. pack on his right shoulder; thus his occupation bears comparison with soldiering. He gave no history of an injury, but for ten days the painful foot had incapacitated him. The X-ray illustration showed: (1) A fracture of the second metatarsal; (2) A mild bulbous enlargement of the shafts of the basal phalanges, second and third toes, and a slight irregularity (? a crack) of the corresponding phalanx of the third toe.

In 1932 F. H. Straus²⁰ reported a case diagnosed radiologically as a sarcoma of the second metatarsal and sent for amputation. His patient was an obese married woman, aged 30, a waitress, the mother of two children. She complained of pain and a hard tumour of the foot, arising without accident, and the pain increased from pain on effort only, until it was continuous day and night; she lost 13 lb. in seven weeks. X-rays showed a swelling of the second metatarsal shaft at the junction of the middle and distal thirds, with hazy and indistinct margins, whilst the medulla was unchanged. No fracture line was made out. The shaft of the third metatarsal showed thickening and increased density. There is apparently a fracture of the shaft of the first phalanx of the fourth toe, but this is probably an artefact. The second metatarsal was excised; the swelling was hard, but dintable by the finger, and an old fracture was obvious on section of the bone. The microscopical section showed well-developed and partially calcified osteoid tissue, there being no inflammatory reaction. Straus observed that the development of callus becomes painful and attracts attention, and offered the warning that it may be mistaken for a neoplasm and amputation considered, unless March Foot is remembered. This is another instance of unduly radical treatment.

DISCUSSION.

According to the modern conception, the mechanics of the human foot depend on adequately strong and properly balanced muscles of the calf and foot. If these are efficient, then, irrespective of the bony architecture and ligaments of the foot concerned, the patient will probably be able to carry on with his or her occupation satisfactorily. On the other hand, Morton²² has shown that certain feet function under mechanical disadvantages, being architecturally weak. He describes four signs or defects which can be diagnosed by X-rays, and states that when they are present the potentiality of anterior foot trouble is considerably increased. The signs are:—

1. Laxity of the joint between the internal cuneiform bones and between them and the scaphoid, resulting in hypermobility of the first metatarsal (*see Figs. 87, 89*). This is shown by a distinct separation between the first and second cuneiforms, resembling a backward continuation of the first inter-metatarsal space (*see Figs. 87, 89*).

2. Shortness of the first metatarsal, this causing over-pronation of the foot, i.e., planus of the anterior part of the foot, as the head of the metatarsal is not long enough to reach the ground in the normal attitude (*see Figs. 81, 89, 90*).

3. Posteriorly located sesamoid bones at the head of the first metatarsal, these representing its point of contact with the ground, and when they are

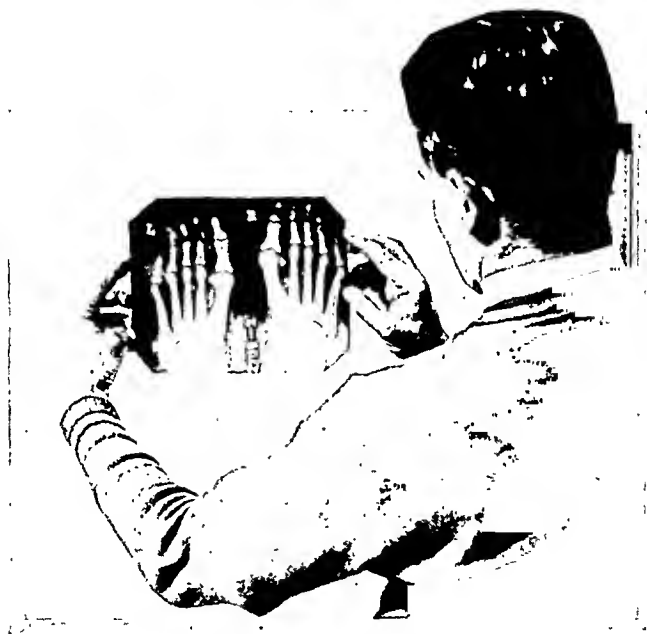


FIG. 90.—“The Ashes winner surveys a part of himself: Larwood with the X-ray photograph of his injured foot.”²³ Note (1) the short left first metatarsal, (2) the thickened shaft of the left second metatarsal.

An architecturally ‘weak’ left foot (Metatarsus atavicus); it gave trouble during the Australian Test Matches.

By kind permission of the “Illustrated Sporting and Dramatic News.”

posterior to the line of the head of the second metatarsal, they have practically the same effect as that of a short metatarsal, i.e., they prevent adequate weight-bearing by the first metatarsal (*see Fig. 88*).

4. Morton points out a further acquired physiological characteristic resulting from these defects; it is an enlargement of the shaft of the second metatarsal bone, especially in its transverse diameter, arising in response to the increased burden thrown on it by the incompetent first metatarsal (*see Figs. 81, 87, 89, 90*).

I have examined, for Morton’s four points, the radiograms of fourteen March Feet illustrated by divers authors, and *Table I* shows their various

Table I.—TABLE SHOWING THE CHARACTERISTICS OF ELEVEN MARCH FEET.

No.	CASE AND LOCATION OF MARCH FOOT	SHORT 1ST METATARSAL	POSTERIORLY PLACED SESAMOID OF 1ST METATARSAL	SPACE BETWEEN 1ST AND 2ND CUNEIFORMS	THICKENED 2ND METATARSAL	THICKENED OUTER BORDER OF 1ST METATARSAL	DIAGNOSIS OF FOOT
1	Deutschländer 1. Right 3rd metatarsal	Yes	Yes	Yes	?	Yes	Short and hypermobile 1st metatarsal. Weak foot
2	Deutschländer 2. Right 2nd metatarsal	Yes	Yes	Yes	Yes	Yes	Short and hypermobile 1st metatarsal. Weak foot
3	Jansen 1. Right 2nd and 3rd metatarsal ..	No	Not clear	Not clear	Yes and 3rd metatarsal	Yes	—
4	Jansen 2. Right 2nd metatarsal Left 2nd metatarsal	No No	Not clear Not clear	Yes Yes	Yes Yes	Yes Yes	Hypermobile 1st metatarsal Hypermobile 1st metatarsal
5	Jansen 3. Right 2nd metatarsal Left 2nd metatarsal	No No	Not clear Not clear	Yes Yes	Yes and 3rd and 4th metatarsal Yes and 3rd and 4th metatarsal	Yes Yes	Hypermobile 1st metatarsal Hypermobile 1st metatarsal
6	Jansen 4. Left 3rd metatarsal	No	Yes	Yes	Yes and 3rd metatarsal	Yes	Hypermobile 1st metatarsal
7	Jansen 5. Right 3rd metatarsal Left 2nd and 3rd metatarsal ..	No No	Yes Yes	Yes Yes	Yes and 3rd metatarsal Yes and 3rd metatarsal	Yes Yes	Hypermobile 1st metatarsal Hypermobile 1st metatarsal
8	Strauss. Left 2nd metatarsal	No	Yes	Yes	Yes	Yes	Hypermobile 1st metatarsal
9	Goldmann. Right 2nd metatarsal	No	Yes	Yes	Yes and 3rd metatarsal	Yes	Hypermobile 1st metatarsal
10	Dodd 1. (Fig. 81.) Right 2nd metatarsal	Yes	Yes	No	Yes and 3rd metatarsal	Yes	Short 1st metatarsal
11	Dodd 2. (Fig. 87.) Right 3rd metatarsal (Illustrated only)	No	Yes	Yes	Yes and 3rd metatarsal	Yes	Hypermobile 1st metatarsal
Totals of 11 feet					13 of 13	14 of 14	12 Hypermobile 1st metatarsals 3 Short 1st metatarsals

characteristics. It reveals : (1) Signs of hypermobility of the first metatarsal in 12 out of 13 feet ; (2) A short first metatarsal in 3 cases ; (3) Posteriorly placed sesamoids in all the cases in which an observation is possible ; (4) A thickened second metatarsal in 13 feet, and a similar affection of the third in 7 cases, and of the fourth in 1 ; (5) Marked increased density of the outer border of the first metatarsal in all cases.

Thus, *Pied Forcé* is most likely to occur in weak feet architecturally.

I believe that *March Foot* is a complication of subacute flat-foot occurring in architecturally weak feet. In these, muscular spasm and exhaustion alternate, and, as the latter supervenes, the stout ligaments of the foot are gradually stretched, and direct trauma to the bony skeleton of the foot occurs. These undamped shocks will produce effects in the weakest bones first, and the slender, resilient metatarsals fall into this category.

As the flat-foot develops, the feet take up the usual flat-foot position, pointing outwards, instead of approximately straight forwards. Thus the body weight is no longer carried through a line passing between the first and second metatarsals, parallel to their shafts, and distributed squarely on to the five heads of the metatarsals, but falls largely in an oblique direction on the inside of the foot, i.e., on the first most (if it is normal), the second next, then the third, and to a lesser degree on the fourth and fifth. If the foot is architecturally weak, as appears to be frequently the case in *March Foot*, then a hypermobile first metatarsal will roll away from this weight, and as a congenitally short metatarsal cannot reach to the ground to carry the strain, then the weight must pass primarily on to the second metatarsal and in decreasing amounts through the third, fourth, and fifth.

Again, the first metatarsal is a much more robust bone than either the second or third, so that structural changes resulting from abnormal strains will be shown much earlier in the latter than the former. Extra stress on the first metatarsal does occur ; this is shown by the thickening and increased density of its outer border, i.e., buttressing, this being the side which would tend to bend concavely. This sign is present in all the fourteen radiograms examined (*see Figs. 81, 87, 88, 89*).

The second metatarsal has the following characteristics : (1) It is usually the longest and slenderest ; (2) It is the most fixed, especially at its base, where it is firmly embraced by the first and third cuneiforms and also by the bases of the first and third metatarsals ; (3) In flat-foot, after the first metatarsal, the brunt of the misplaced weight falls on it.

The next most commonly affected bone, the third metatarsal, also has these characteristics to a rather lesser degree, i.e., it is slender and long, and is more fixed than the first, fourth, or fifth metatarsals.

These points, I think, explain why, when the supporting power of the muscles and ligaments of the calf and foot is exhausted, the second metatarsal will be the first to 'crack' ('crack' probably describes better than 'fracture' what actually occurs), and why the third is next affected.

March Foot is probably an auto-traumatic complication of subacute flat-foot in an architecturally weak foot, rather than a separate clinical entity. The different diagnoses suggested by the many authors who have described it are varying manifestations of the inclusive condition of subacute

flat-foot, e.g., tenosynovitis, spasm of museles, periostitis, synovitis, arthritis, rheumatism, and finally fracture with callus formation, and do, in turn, at one time or other, play a part in the formation of the picture discussed.

DESCRIPTIVE SUMMARY OF MARCH FOOT.

Etiology.—March Foot is a subacute painful affection of the fore part of the foot, occurring chiefly about the shafts of the metatarsals and the surrounding soft parts. It develops insidiously with slowly increasing pain, which at first arises after prolonged excessive effort, later after ordinary exercise, and ultimately becomes continuous and incapacitates its victim.

It appears to be a condition which is becoming rarer; twenty to forty years ago reports of cases in batches of 15 to 40 were common, but during the last ten years the reports are of much smaller numbers, e.g., Deutschländer¹³ 6, Jansen¹⁷ 6, Goldman¹⁹ 1, Straus²⁰ 1, Dodd 1.

The majority of cases occur in soldiers carrying full packs on active service, although civilians engaged in similar heavy occupations are affected, e.g., a pedlar. With the present-day ample mechanical transport facilities, hard foot-slogging in civilian life is much rarer, whilst periodically in the daily papers appear pictures of the 'mechanized army', so that its incidence in military or civil circles is scarcely likely to be on the increase.

The great military nations, Germany and France, have produced the bulk of the cases and the literature thereon. Doubtless much of their conscript material would not be ideal for the hard soldiering conditions of thirty years ago.

Women with arduous standing and weight-carrying work (e.g., waitresses, shop-assistants, and nurses) also develop the condition.

Clinical Features.—The swelling appears on the dorsum of the foot, centred usually about the shafts of the second and third metatarsals, invading the soft tissues and bone. It scarcely pits on pressure, is slightly reddened, and is tender. A bony swelling of the shaft of one of the metatarsals, usually the second or third, becomes palpable several weeks later. This is callus, which has arisen usually around an oblique or v-shaped fracture of the metatarsal shaft, at the junction of the middle and distal thirds. This fracture occurs quietly without noticeable incident as the condition develops; it is not an exciting factor, and is not always present. The callus may be mistaken for a new growth, suggesting amputation unless March Foot is borne in mind (see Fig. 81). On careful elicitation, crepitus may be detected at the site of the swelling.

There are, in addition, signs of a flat-foot with callosities, and spasm of the interossei, denoted by fixity of, and pain on attempts to move sideways, the second or third toes, is usually present.

X-ray Findings.—These are of clinical and historical interest, as March Foot was probably amongst the first conditions to be investigated by radiology, records being available thirty-four years ago. In the fully developed case. X-rays show:—

1. A bony swelling of somewhat fluffy, bulbous outline, due to callus, at the junction of the distal and middle thirds of either, and occasionally even of

both, the shafts of the second or third metatarsals, much less often of the fourth or fifth, and extremely rarely of the first. This swelling is around a partial or complete fracture, usually without displacement. As recovery progresses, it becomes smaller and more sharply defined.

2. In the early stages there is increased density of the shafts of the metatarsals where the interosseous muscles arise, i.e., the second, third, fourth, and inner border of the fifth; the outer border of the first metatarsal shaft is also dense, but the change is most marked in the second or third metatarsal shafts (*see Figs. 81, 87*).

3. According to Jansen, other bulbous swellings may arise about the shafts of the metatarsals, and I have observed slight ones about the shafts of the first phalanges of the second, third, and fourth toes (*see Figs. 81, 87-89*). These are probably due to localized periostitis at the site of attachment of the flexor tendon-sheaths.

4. Of fourteen radiographs of cases illustrated by various authors, all show signs of weak architecture. (This point has already been discussed.)

Pathology.—The bony swelling is firm, pinkish white, and microscopically consists of calcified osteoid tissue with periostitis (Dodd and Straus). It forms at the junction of the middle and distal thirds, this point being the site of entry of the nutrient blood-vessels (Deutschländer¹⁴). (*See Fig. 85*.)

Diagnosis.—This is suggested by: (1) The patient's occupation; (2) The history of incapacitation due to pain in the fore part of one or both feet; (3) The presence of a swelling partly of the soft tissues and also of the bone about the metatarsal shafts; (4) Occasionally crepitus detected in the metatarsal, which is the seat of the swelling; (5) The X-ray picture; (6) The progressive recovery which follows rest, adequate support, and care of the feet.

In the differential diagnosis arises the question of a sarcoma of the shaft of the metatarsal; the possibility of a syphilitic periostitis, which is excluded by the Wassermann reaction; and a low-grade hæmatogenous osteo-periostitis (Deutschländer) and arthritis in the metatarso-phalangeal joints, but when this is present patients are unable to stand on their toes owing to pain.

TREATMENT.

This consists in:—

1. Rest in bed until the pain and œdema subside, with complete immobilization of the foot or feet by plaster, which is applied to maintain a dorsiflexed and an inverted position with a well moulded arch. If necessary, manipulation of the feet under anæsthetic into this over-corrected position is advisable.

2. With the subsidence of pain, patients get up, and are carefully fitted with stout shoes or boots which adequately support the feet. This foot-wear is adjusted with inside wedges to the heel and sole, metatarsal bars, or even an outside iron with an inside T-strap if necessary.

Patients are carefully instructed in the toilet and care of the feet, a card with the following italicized points printed on it being given to them.

a. Scrub the feet and legs daily in hot water with a soft brush or loofah glove. This ensures a form of homely and effective massage to the feet and muscles of the calf; the actual bending is also beneficial to the abdomen.

b. Wear thick stockings or socks, and change these frequently. These must be large enough, not bunching the toes together.

c. Avoid standing. Standing strains the already weakened calf muscles calling for a sustained muscular effort, whilst walking requires an effort alternating with momentary rests.

d. Walk with the toes pointing directly forwards, never turned outwards. This tends to throw the weight squarely on the forefoot, that is. away from the strained inside arches, and checks lateral stresses.

e. Shoes or boots must be worn from the moment of getting out of bed, until getting into bed at night. This ensures arch support all day.

f. Never walk in soft slippers or in stocking feet. This avoids unsupported arches.

g. When sitting, the feet should be put up on a chair or couch, if possible. This permits an easy venous return and removes the weight of the legs and thighs from the feet (this is considerable, as is readily realized when carrying an amputated limb, or holding a limb during operation.) Whilst sitting thus, patients may remove their boots, but must put them on before moving.

h. Practise moving the feet and toes up and down about twelve times before or after food daily, also when in bed, on the bus, or on the train. This retones the muscles of the feet and calves, and is definitely helpful, although it appears to be a trivial exercise. Later, walking on tip-toes, and particularly on the heels, should be practised. I regard the latter as the best flat-foot exercise, although it is admittedly difficult.

3. Graduated exercises of the feet and legs are persisted in, to redevelop the lost muscular tone, with the slow resumption of the usual occupation until the muscle power is equal to all ordinary and extraordinary calls likely to be made on it.

4. Obesity, varicose veins, visceroptosis, general muscle flabbiness, and poor bodily carriage are treated, whilst septic foci (teeth and tonsils, etc.) whose toxins diminish muscle tone are searched for, and, if possible, removed.

Finally, in view of the permanent, architectural weakness of these feet, patients must be warned that more consideration of their feet than is usual will always be necessary and that sound, well-fitting footwear must be worn.

I wish to thank my radiological colleague, Dr. R. Parry, for his help in the interpretation of the X-rays he has so kindly lent me to illustrate this article, and also the *Illustrated Sporting and Dramatic News* for the loan of the block of Fig. 90.

REFERENCES.

¹ BREITHAUPT, "Zur Pathologie des menschlichen Fusses", *Med. Zeits.*, 1855. (Quoted from Kirschner.)

² PAUZAT, "De la Périostite ostéoplastique des Metatarsiens à la Suite de la Marche", *Arch. de Méd. et Pharm. mil.*, 1887, 337.

³ WEISBACH, *Deut. mil.-ärztl. Zeits.*, 1877, No. 12.

- ⁴ POULET, "De l'Ostéopériostite rhumatismale des Metatarsiens", *Arch. de Méd. et Pharm. mil.*, 1888, 254.
- ⁵ MARTIN, "Inflammation périostite-arthritique du Pied de la Suite des Marches", *Arch. de Méd. et de Pharm. mil.*, 1891, 327.
- ⁶ BUSQUET, *Rev. de Chir.*, 1897. (Quoted from Boisson and Clapotot.)
- ⁷ SCHULTZ, "Die sogenannte Fussgeschwulst", *Arch. f. klin. Chir.*, 1897, iv, 872.
- ⁸ STECHOW, "Fussödem und Röntgenstrahlen", *Deut. mil.-ärztl. Zeits.*, 1897, 465.
- ⁹ CHAPOTOT AND BOISSON, "Le Pied forcé: Étude sur la Nature, et la Pathogénie des Lésions de l'Avant-pied, provoquées par la Marche chez les Fantassins", *Arch. de Méd. et de Pharm. mil.*, 1899.
- ¹⁰ THIELE, *Deut. med. Woch.*, 1899, xxv, 158.
- ¹¹ MOMBURG, "Die Entstehungsursache der Fussgeschwulst", *Deut. Zeits. f. Chir.*, 1904, lxxiii, 425.
- ¹² KIRSCHNER, "Die Aetiologie der indirecten metatarsal Fracturen", *Arch. f. klin. Chir.*, 1905, lxxvii, 241.
- ¹³ BÄHR, "Die Fussgeschwulst und ihre Beziehungen zum anderen Gewolbe", *Zeits. f. orthop. Chir.*, 1913, xxxii, 312.
- ¹⁴ DEUTSCHLÄNDER, "Ueber entzündliche Mittelfuss Geschwulst", *Arch. f. klin. Chir.*, 1921, cxviii, 530.
- ¹⁵ JONES, SIR ROBERT, *Orthopaedic Surgery of Injuries*, 1921.
- ¹⁶ JONES AND LOVETT, *Orthopaedic Surgery*, 1923.
- ¹⁷ JANSEN, "March Foot", *Jour. Bone and Joint Surg.*, 1926, viii, 262.
- ¹⁸ GRAY, *Anatomy*, 21st ed., 556.
- ¹⁹ GOLDMAN, "March Foot, with Fracture of Metatarsal Bone", *Jour. Bone and Joint Surg.*, 1928, x, 228.
- ²⁰ STRAUS, *Surg. Gynecol. and Obst.*, 1932, liv, No. 3, 581.
- ²¹ *N. Y. Med. Jour.*, 1899, Editorial.
- ²² MORTON, *Amer. Jour. Surg.*, 1930, ix, 315.
- ²³ *Illustrated Sporting and Dramatic News*, 1933, April 22, 178.

*SHORT NOTES OF
RARE OR OBSCURE CASES*

REPEATED PERFORATION OF PEPTIC ULCERS.

By H. B. BUTLER,

SURGEON, ROYAL SURREY COUNTY HOSPITAL, GUILDFORD.

A. W., aged 28 years, was admitted nine years ago with symptoms of duodenal ulcer. Appendicectomy was performed and medical treatment instituted. Two years later he was admitted with perforated duodenal ulcer; the perforation was closed by operation. Nine months later still he was admitted with pyloric obstruction, and a posterior gastrojejunostomy was performed.

On June 4, 1931, he was admitted with perforated jejunal ulcer. The gastrojejunostomy was undone, the stomach and transverse mesocolon were repaired, and the portion of the jejunum containing the ulcer was excised, so that the original anatomy was restored. The pylorus was examined, and, though evidently contracted, it was hoped that it might prove adequate, as the patient had had enough.

He did well for three weeks after this operation, but then began to vomit, at first a little, but gradually increasing until no food was retained. On July 28, the patient being very ill, the abdomen was again opened and an anterior gastrojejunostomy was performed as rapidly as possible.

The patient made an uneventful recovery from the last operation, and remained well until fourteen days before his readmission on Oct. 2, 1932. He was vomiting and had a tender lump in the abdomen, 2 in. above the umbilicus and under the left rectus. This was correctly diagnosed to be a jejunal ulcer and he was put on Hurst's ulcer diet, under which treatment he improved considerably. It was decided to remove a large portion of his stomach in the hope of producing a permanent achlorhydria to prevent the formation of further ulcers.

At operation on Oct. 11 the jejunal ulcer was found perforated into the anterior abdominal wall and there attached. The ulcer was detached and the gastrojejunostomy was undone. This part of the operation was made difficult and tedious by adhesions. The damaged jejunum was resected. The hole in the anterior wall of the stomach was not repaired, but a partial gastrectomy of the Pólya type was proceeded with—the pylorus was found to be completely occluded. The portion of the jejunum used for the anastomosis was just distal to the resected portion and it was brought up anterior to the transverse colon. A gastric ulcer on the lesser curve, hitherto unsuspected, was found in the portion of stomach removed.

The patient made an uninterrupted recovery, and remains quite well and looks extremely fit. Gastric analysis charts (*Figs. 91, 92*) taken before and after operation show that the desired result, i.e., achlorhydria, apparently has been attained.

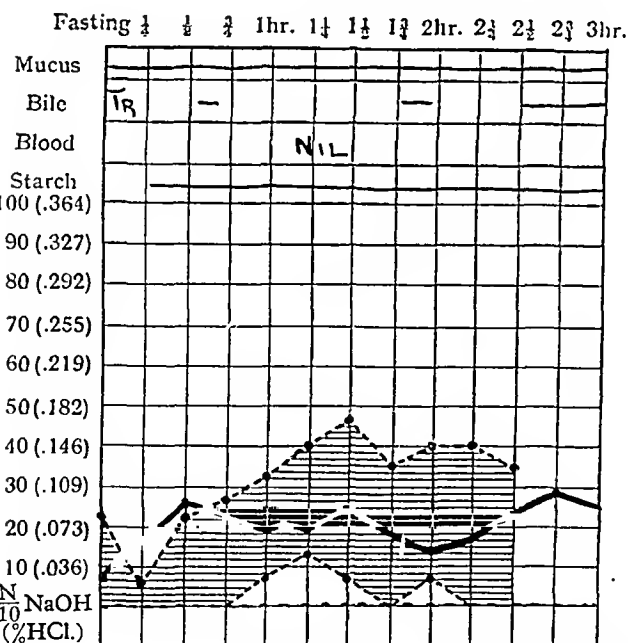
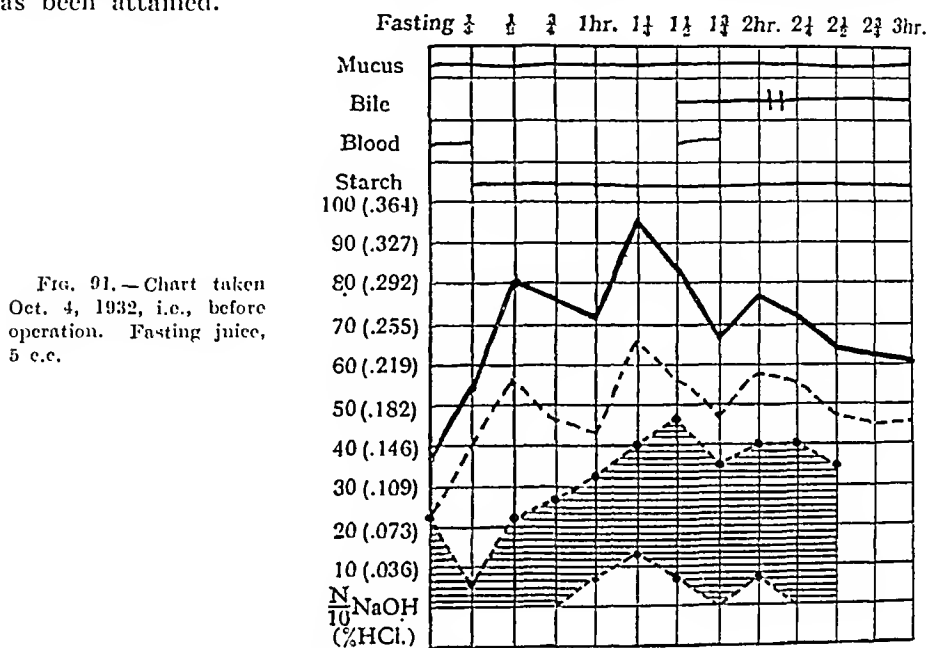


FIG. 92.—Chart taken Dec. 10, 1932, showing complete achlorhydria following gastrectomy. Fasting juice, 12 c.c.

The shaded area represents the limits for free HCl in 80% of normal people, and average rate of emptying (2–2 $\frac{1}{2}$ hours). Broken line represents free HCl. Continuous line represents total acidity.

PEDUNCULATED HYDATID CYST OF LIVER.

BY N. ROSS SMITH, BOURNEMOUTH.

THE following case is recorded because of the rarity of the disease in England and the obscurity of the diagnosis.



FIG. 93.—Skiagram showing calcified hydatid cyst in abdominal cavity. The pedicle attaching the cyst to the lower border of the liver can be seen. (*Skiagram by Dr. D. D. Malpas.*)

HISTORY.—Mrs. H., a lady aged 71 years, gave a history of vague 'indigestion' and attacks of right-sided abdominal pain extending over a period of more than twenty years. The attacks of pain had occurred at intervals of several months up to a year and had lasted from a few hours to a day or two. They had been attributed to 'gall-bladder trouble' and had

subsided without operative treatment. Three weeks before operation she had become jaundiced without pain and had developed intermittent pyrexia of moderate degree. She had lost one stone in weight in the previous six months. All her life had been spent in England, except for a visit to Algiers twenty-four years previously and several visits since then to the French Riviera.

ON EXAMINATION.—The patient was somewhat wasted and moderately deeply jaundiced. In the abdomen to the right of and below the umbilicus was palpable a slightly movable, firm, kidney-shaped mass. This appeared to be distinct from the liver, which was uniformly enlarged downwards to one inch below the costal margin. The faeces were brown in colour, and tests for bile in the urine were negative. Blood-count: Leucocytes, 22,000; polymorphs, 87.5 per cent; lymphocytes, 5 per cent; mononuclears, 7 per cent; eosinophils, 0.5 per cent. Blood-urea and urea-concentration tests were normal. In a cholecystogram (oral method) no shadow appeared in the region of the gall-bladder, but a shadow of unequal density was seen on the right side, the shape and position of the mass discovered on physical examination (*Fig. 93*). This shadow was thought by the radiologist to be possibly due to accumulation of dye in the caecum. As hydatid disease was not suspected, no special tests were carried out. A provisional diagnosis was made of carcinoma of the caecum with metastases in the liver.

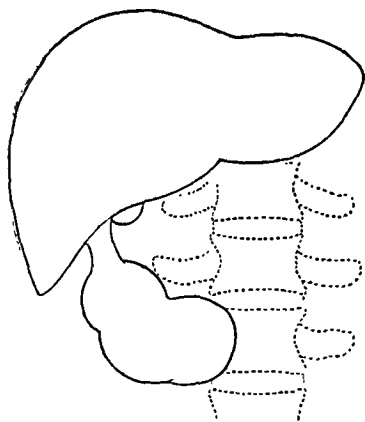


FIG. 94.—Diagram showing size and position of the cyst relative to the liver.

OPERATION.—At operation the greater omentum was found adherent to the liver and duodenum, obscuring the gall-bladder and ducts, and was wrapped around and toughly adherent to the tumour felt before operation. The omental adhesions were freed and it was then seen that the tumour was hanging from the lower border of the right lobe of the liver by a pedicle of liver tissue, $1\frac{1}{2}$ in. wide, $\frac{1}{4}$ in. thick, and 1 in. long (*Fig. 94*). This pedicle was ligated and divided and the tumour removed entire. The gall-bladder and ducts were then exposed. The common and hepatic bile-ducts were found greatly dilated, being obstructed by a stone in the ampulla of Vater, which

was removed after opening the common duct above the duodenum. The gall-bladder appeared healthy and was not removed. Convalescence was uneventful.

PATHOLOGICAL REPORT.—The specimen (*Figs. 95, 96*) was sent to the Museum of the Royal College of Surgeons, and Mr. T. W. P. Lawrence kindly reported as follows: "A degenerated and shrunken hydatid cyst. It consists of a main portion, irregularly ovoidal in shape and measuring 4 in. in length and $2\frac{1}{2}$ in. at its widest part and having a well-marked transverse groove at about its middle. Projecting from one side near the lower end is a hemispherical cyst measuring 2 in. in width. At the

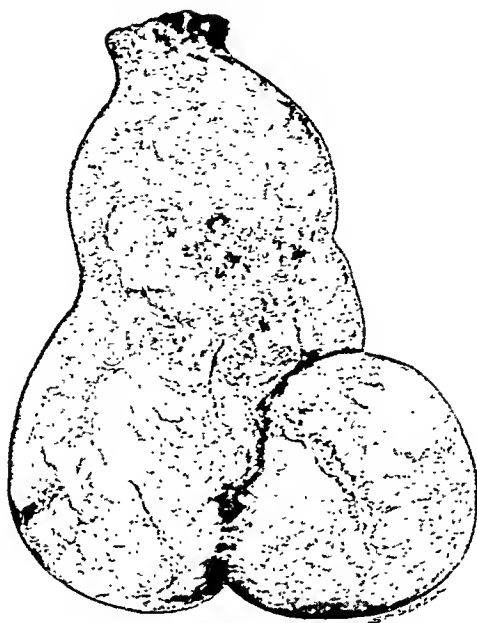


FIG. 95.—Drawing of cyst after removal at operation, showing pedicle, areas of calcification, and remains of adherent omentum.

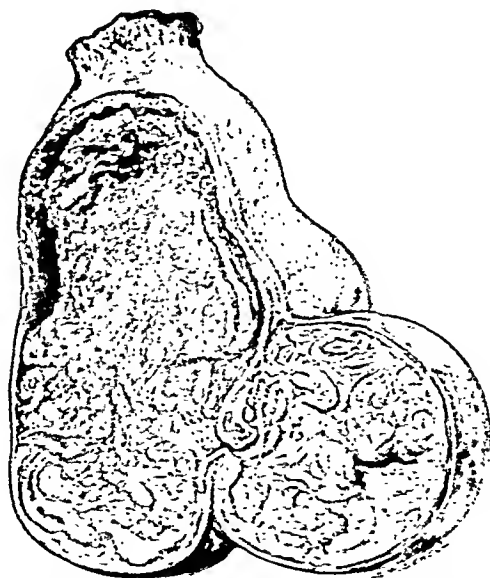


FIG. 96.—Drawing to show the convoluted laminated membrane and granular material in the interior of the degenerated cyst.

other end of the specimen is a raw surface, $1\frac{1}{2}$ in. in diameter, marking the attachment to the liver: in the centre of this several vessels are seen entering the cyst wall. The cyst is completely filled with a gelatinous substance, largely intermixed with mortar-like material; and in the smaller, projecting cyst the contents have partly the form of convoluted, semi-transparent membrane. In the neighbourhood of the pedicle both the contents and the cyst wall are extensively calcified, and in lesser degree calcification affects many other parts of the cyst wall. The cavity of the

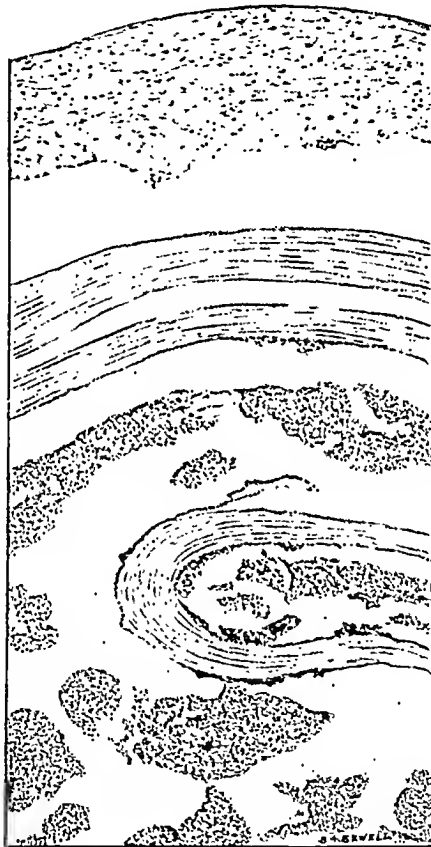


FIG. 97.—Microscopic section, showing adventitia, laminated, and germinal layers. ($\times 20$.)

cyst is single, but the main part only communicates with the smaller hemispherical portion by means of an aperture of about $\frac{1}{8}$ in. in width. Small portions of omental tissue adhere to the external surface of the cyst. Microscopical examination shows a large amount of granular material, together with sections of portions of the proper wall of a hydatid, exhibiting the characteristic parallel fine striations (*Fig. 97*)."

It seems probable that infection with the parasite in this case was acquired not in England, but in Algiers, where hydatid disease is common.

A CASE OF HYDRONEPHROSIS.

BY C. H. FAGGE, LONDON.

MRS. L., aged 53, has for some years complained of indigestion and sickness. Her attacks usually come on at night, and for the last three months have been more severe; they have no relation to meals or diet. She has fairly constant pain or tired feeling in the back. She has noticed her urine has a high smell, and for some years has got up twice in the night to pass water; for the last four months this has increased, so that she is now getting up about four times each night.

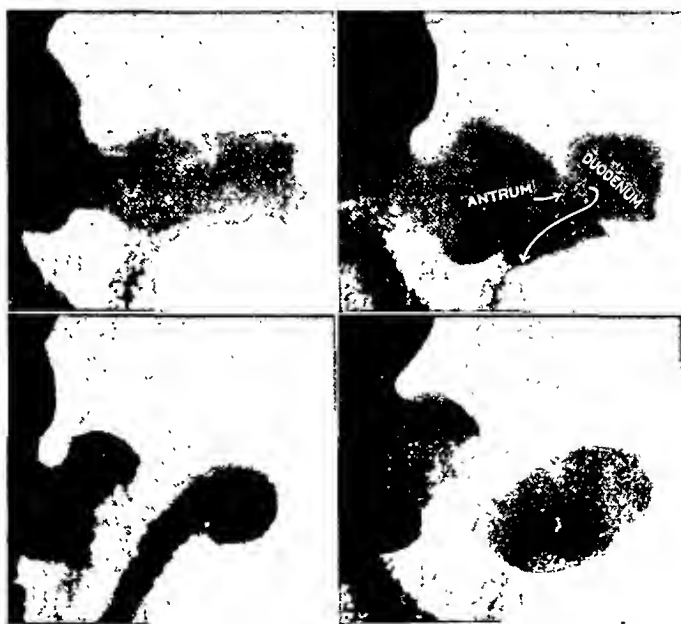


FIG. 98.—Serial view of duodenum taken in the posterior prone position, showing the abnormal position of the second part of the duodenum.

For several years she has found that lying in bed on either side (especially the left) caused heartburn and vomiting; to overcome this she acquired the habit of sleeping flat on her back.

She went to Dr. Rose, of Wimbledon, for the investigation and treatment of her indigestion, and at his suggestion a barium meal was given by Dr. Sparks, who reported as follows:—

“The examination showed a regular contour of the stomach, which was of the normal size with a moderate peristaltic activity and a pronounced convexity of the lesser curvature: the meal passed freely into the first part of the duodenum, which filled well and was globular and regular in outline.

"The second part of the duodenum passed much more medially than usual and showed a well-marked convexity towards the mid-line as if there were some structure displacing it inwards. The serial view of the duodenum (*Fig. 98*) shows this displacement in its upper half. The three-hour radiogram (*Fig. 99*) shows the second part of the duodenum displaced so that the apex of the curve passes just beyond the mid-line of the spine; this view also shows an absence of filled coils of small bowel in the right lumbar region.



FIG. 99.—Anterior view of the abdomen, taken three hours after the meal with the patient supine, showing the medial displacement of the second part of the duodenum.

"As a result of this examination it was felt that the most probable structure to cause this displacement of the duodenum would be the right kidney. A uroselectan examination was made (*Fig. 100*) and showed the right kidney to be lying low in position and to be enlarged to nearly three times the normal size. Multiple dilated calices became outlined with the dye, and no outline of the pelvis or ureter could be distinguished. The left kidney was seen to lie in the normal position and to be functioning

normally. There was seen to be a scoliosis of the lumbar spine with the convexity towards the left."

Though Dr. Sparks suggested that the right kidney was enlarged to three times its normal size, no definite tumour could be felt on physical examination; but there was a sense of soft resistance in the right loin, which was obviously fuller than the left.

OPERATION.—On Dec. 10, 1932, through a right lumbar incision of the Mayo type, i.e., a vertical along the outer border of the erector spinæ continued forward below almost at a right angle, the outer border of the quadratus lumborum was exposed by dividing the lumbar aponeurosis, and a much enlarged kidney was seen. When it was freed from the perinephric fat, to which it was firmly adherent, it was seen that the greater part of the renal tumour was due to a dilated pelvis. The actual kidney substance was much thinned, being not more than $\frac{1}{4}$ in. thick; the much dilated pelvis ending below in a normal ureter. Anteriorly the junction of the two was crossed by an aberrant vessel of moderate size, apparently a tributary of the renal vein. When this was divided the hydronephrosis did not empty itself. The ureter was ligatured and divided, and the isolated renal vessels were dealt with in the same way. The wound was closed with drainage. The patient made a good recovery: since the operation she can sleep without discomfort on either side and has lost all gastric symptoms.

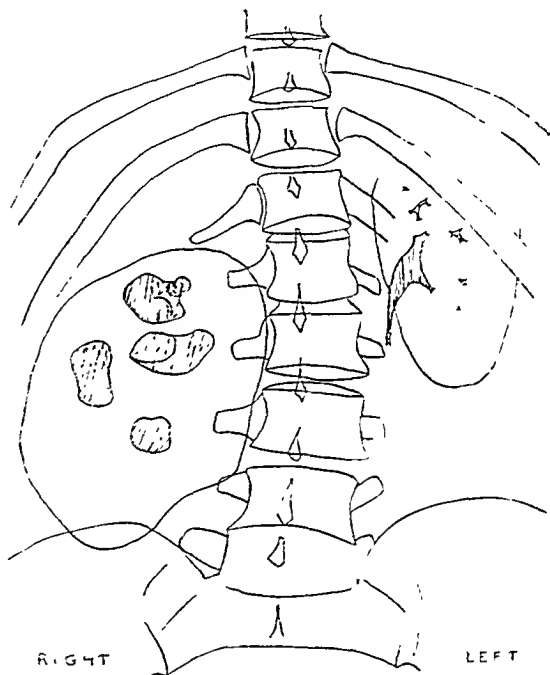


FIG. 100.—Outline tracing of film made twenty minutes after injection of uroselectan.

COMMENT.

The case appears worthy of record because an extreme degree of hydronephrosis had occurred without any urinary symptoms. The location of the disease to the correct organ was, in fact, entirely due to the X-ray report, and we have no knowledge of any previous case in which a renal swelling has so interfered with the passage of substances through the duodenum as to give rise to the appearance such as is seen in the barium X-ray examination. In view of this partial obstruction to the duodenum it is reasonable to suppose that the patient's gastric symptoms were due to the renal tumour, and this

was supported by the fact that since removal of the enlarged kidney she has had no recurrence of these attacks.

After removal from the body it is easy to demonstrate the definite thickening of the ureter at the pelvi-ureteral junction and the valvular type which is usually regarded as due to some congenital error of development, though the question will naturally arise whether this thickening may be caused by fibrosis secondary to ureteral obstruction by the vessels which passed, if not exactly over the point of narrowing, certainly in close proximity to it.

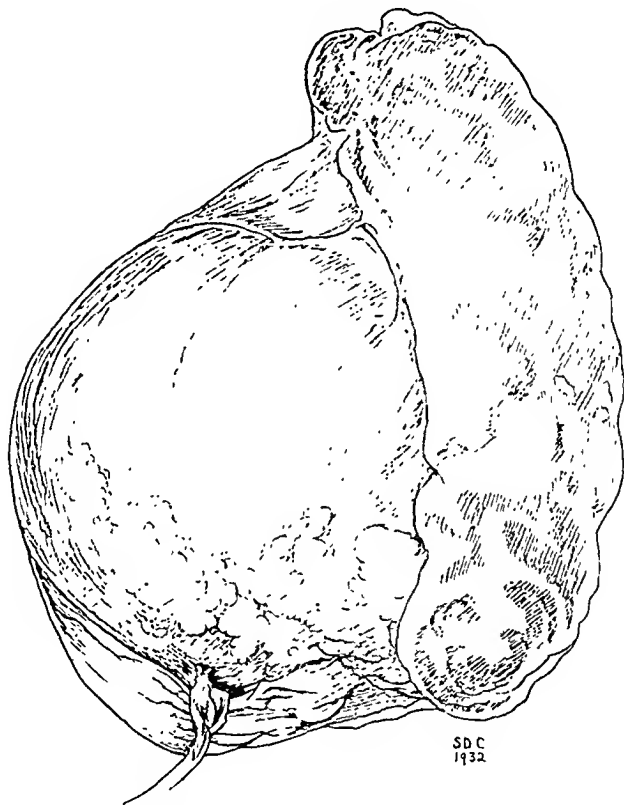


FIG. 101.—Posterior aspect of kidney. ($\times \frac{1}{2}$.)

We have never been able to convince ourselves that a soft vessel-like vein can obstruct the thick-walled ureter; it appears to us that the main factor in hydronephrosis attributed to abnormal vessels is associated abnormality of the kidney. There is no evidence that this patient's kidney ever was unduly mobile, and, on the contrary, at the time of operation it was found to be unusually fixed from adherence of its capsule to the perinephric fat.

The drawing of the specimen (*Fig. 101*) shows clearly that the displacement of the duodenum was due to a 'pelvic' hydronephrosis: probably this is less common than the renal type, and, if so, an explanation is afforded of the suggestion that such a radiological appearance has not been previously recorded.

A DIVERTICULUM OF THE FIRST PART OF THE DUODENUM.

By G. E. GASK,

SURGICAL UNIT, ST. BARTHOLOMEW'S HOSPITAL.

Duodenal diverticula have become well known since the advent of X rays and the use of the opaque meal as a routine method of examination of gastric cases. The large majority of these pouches are found in the second part of the duodenum, and though anatomists, noticeably J. C. Grant,¹ have recorded pouches arising from the first part, the details of a case which was operated on seem to merit a record.

The patient was a man, aged 37, with a history suggesting duodenal ulcer. One day he had a sudden severe attack of abdominal pain, suggesting a perforation, which brought him to hospital.

The X-ray diagnosis was a duodenal ulcer and a diverticulum, which filled and emptied easily from the first part of the duodenum (*Fig. 102*). At operation this condition was confirmed. There was an ulcer of the duodenum which had perforated and been sealed off by peritoneal adhesions, and there was a pouch arising from the lower border of the duodenum just distal to the pylorus and proximal to the ulcer. The pouch was about $1\frac{1}{2}$ in. long by 1 in. in diameter, and



FIG. 102.—Radiogram showing a duodenal ulcer and a diverticulum.

the opening into the duodenum was large enough to admit the tip of the finger. It was soft and thick, resembling the stomach in texture.

It was considered that the pouch was of congenital origin, there being no evidence to suggest that it had formed as the result of pressure.

The pouch was excised and a gastrojejunostomy performed. There is nothing to note concerning the after-history of the patient. Histological examination showed normal duodenal structure.

REFERENCE.

¹ GRANT, J. C., *Jour. of Anat.*, 1923. lvii. 357.

A CASE OF CARCINOMA OF THE RIGHT BREAST FOLLOWED BY SARCOMA OF THE LEFT BREAST AFTER AN INTERVAL OF EIGHTEEN YEARS.

By CECIL P. G. WAKELEY,
SURGEON TO KING'S COLLEGE HOSPITAL.

THE following case is of interest because the patient suffered from a scirrhus carcinoma of the right breast which was removed in 1914, and was not followed by a recurrence or secondary deposits. After an interval of eighteen years, at the age of 89, a hard tumour was discovered in the left breast and when the breast was amputated it was found to be a chondrosarcoma.



FIG. 103.—Microscopic section of right breast, showing typical spheroidal-celled carcinoma. ($\times 130$.)

The clinical history is as follows:—

Mrs. A. M. B., age 71, was operated upon in 1914 for a tumour in the right breast. The patient had noticed the lump for just over a year. The tumour was hard, about the size of a hen's egg, and situated in the upper and outer quadrant of the breast; it was attached to the skin, but not to the pectoral fascia. There were a few hard glands in the axilla. Although the patient was 71 years of age her general condition was good, and a radical removal of the breast was advised. The operation was

performed as completely as possible; both pectoral muscles were removed with the breast and all the fat and glands in the axilla. The patient made a rapid recovery and was able to return to her home within three weeks of the operation.

Microscopical examination of the tumour of the breast proved it to be a spheroidal-celled carcinoma of the scirrhus type. The glands and fat removed from the axilla did not reveal any carcinomatous invasion when examined microscopically.

My colleague, Dr. Creed, reported as follows on the microscopical section of the tumour: "The section shows a spheroidal-celled carcinoma (*Fig. 103*). The tumour cells are arranged in masses of moderate size with no definite lumen formation. Small strands, a single cell thick, can be seen invading the stroma. Stroma and tumour masses are present in approximately equal proportions. The stroma is composed of fibrous tissue of moderate density, and shows no cellular infiltration with lymphocytes or plasma cells."



FIG. 104.—Microscopic section of left breast, showing chondrosarcoma. ($\times 130$.)

The patient remained very well until June, 1932, when a hard lump was noticed in the left breast. She was 89 years of age and somewhat senile and infirm, and could not say how long she had known the tumour to be present. The tumour was about the size of an orange, it was hard and irregular to the touch, but was not attached to the skin or to the underlying structures. There were no enlarged glands in the axilla.

Local removal of the breast was decided upon because it was thought that the tumour might eventually fungate through the skin. A radical

amputation was considered too severe an operation for a patient of her age and in a feeble condition.

On June 22, 1932, a local amputation of the breast was performed under gas and oxygen anaesthesia. The patient stood the operation well and convalescence was uninterrupted. The wound healed soundly. The tumour was hard and occupied practically the whole of the small senile breast.

Dr. Creed examined the tumour and reported as follows: "The sections show a chondrosarcoma (*Fig. 104*). There are some areas which show fairly typically the appearance of a spindle-celled sarcoma with some deposition of collagen fibrils and with active mitosis. Most of the tumour, however, is chondromatous in structure with cells resembling cartilage cells embedded in a matrix which varies in appearance. Some parts of the matrix are hyaline and eosinophil, and others are fibrillary and tend to be basophil. Some of the tumour is necrotic. The spindle-celled areas are principally towards the periphery of the tumour but merge without any sharp line of demarcation with the chondromatous areas."

The patient was examined in March, 1933, and is in good health with no signs of recurrence.

CASE OF HERNIA INTO THE FOSSA INTERSIGMOIDEA.

By RONALD REID, COLCHESTER.

CASES of retroperitoneal hernia in the fossa intersigmoidea are rare; two have been reported in detail. The first is the case described by Eve in the year 1885, the second was recorded by McAdam Eccles in 1895.

The intersigmoid fossa was first mentioned in 1742 by Hensing, and then in 1769 by Haen, by Roser in 1843, Treitz in 1857, and also by Waldeyer in 1868. It is present in the majority of subjects—according to Treves in 52 per cent. Moynihan found the fossa in 70 per cent, Waldeyer in 84 per cent, of cases examined. The opening of this fossa is found by turning the pelvic mesocolon over to the right, and it is then seen as an oval orifice at the root of the mesentery. The long axis of the oval is transverse and measures about $1\frac{1}{2}$ in. The fossa in the adult varies from a small depression up to the size of a hen's egg. One case of a much larger fossa has been described; here the fundus reached to the aortic bifurcation. The opening lies over the left psoas muscle, and is bounded in front by a sharp peritoneal fold; below and behind the boundary is the smooth parietal peritoneum sweeping up into the fossa. The long axis of the fossa is directed from below upwards and to the right, the fundus usually reaches the bifurcation of the common iliac vessels in a well-marked specimen, and in the posterior wall of the pouch is the ureter.

The following case report is of interest by reason of the rarity of this form of hernia. It appears that this hernia is not discovered apart from acute intestinal obstruction.

The patient, an old woman aged 69 years, was admitted to hospital in February, 1931. Ten days before admission she had noticed mild colicky

pains in the lower part of the abdomen. After two days the pains became worse and vomiting occurred; the pains continued and there was an action of the bowels on the following day. There was an action again on the next day, but for the five days previous to admission there was absolute constipation. Two days before admission faecal vomiting started and the pains became intense and almost continuous; they were still colicky in nature, and greatest to the left of the lower abdomen. An enema was given, but it failed to give relief.

The past history contained nothing relevant to the present condition.

ON ADMISSION.—The general condition of the patient was poor in the extreme. The expression did not indicate severe toxæmia, but the pulse was feeble and thready, the rate being 108; the temperature was subnormal. The abdomen was grossly distended, chiefly in the hypogastrium, and was tympanitic on percussion. No intestinal movements were seen or heard. Tenderness was general, but as the left iliac fossa was approached the pain on deep palpation was very severe. On rectal examination general abdominal pain was produced, and nothing abnormal could be felt. The urine was normal.

A diagnosis of acute intestinal obstruction was made. The descending or pelvic colon was thought to be the site of the obstruction and its nature carcinomatous.

OPERATION.—The patient was given $\frac{1}{4}$ gr. of morphia and taken to the theatre. It was found that the general condition was deteriorating very rapidly, and it was therefore decided that the only chance of saving the patient was by relieving the obstruction as quickly as possible through a blind cæcostomy. Accordingly a local anæsthetic was given and the abdomen opened by a grid incision over the cæcum. At once a distended coil of small intestine pushed through the incision, and the collapsed cæcum was discovered. The small intestine was so dilated and engorged that it was only after increasing the original incision that the site of obstruction was found in the left iliac fossa. The exact nature of the obstruction could not be made out, the bowel could not be freed, and the patient was almost *in extremis*. No further time was lost, the coil of distended bowel next to the obstruction was clamped and very rapidly anastomosed to the coil of collapsed intestine found below the obstruction; the anastomosis was stitched to the parietal wound and a soft rubber catheter was inserted into the upper coil, just above the site of anastomosis. The abdominal wound was closed with free drainage: 40 c.c. of anti-gas (*B. welchii*) serum were given intramuscularly.

The state of the patient began to improve after four hours. vomiting ceased, and the discomfort diminished. Distension was less, although there was only moderate drainage from the enterostomy. Glucose rectal saline (6 per cent) was ordered four-hourly; the patient could retain only 4 oz., and after twenty-four hours could retain practically none. It was decided that fluid must be put into the circulation by the intravenous route. The serum was repeated and 500 c.c. of gum saline were run into the right median basilic vein, fifty minutes being occupied with this operation. At the end of the injection the patient complained of pain in the left hand, then in the

arm, and finally in the chest behind the sternum. There was shortness of breath, and gradually the patient became unconscious, sank slowly, and died about thirty-four hours after the time of the laparotomy.

POST-MORTEM.—The abdomen contained a few coils of distended and thickened small intestine and the remainder of the small and large intestine, collapsed and to all appearances normal. There was an obstruction of the jejunum about four feet from the duodenojejunal flexure where the intestine entered a pouch on the lower aspect of the pelvic mesocolon. The jejunum entering the pouch was thickened, and appeared to be hypertrophied, as though the obstruction was of long standing. With little difficulty the loop of jejunum was withdrawn from the pouch: there were some adhesions, and considerable œdema of the loop and of the pelvic mesocolon adjacent to the hernial opening. The loop withdrawn was about 5 in. in length, dark purple in colour, and showed a patch of gangrene at the site of constriction of the entering limb.

The opening of the fossa into which the bowel had entered was on the lower and left-hand aspect of the pelvic mesocolon, at the parietal border. The shape was oval, measuring about $1\frac{1}{2}$ in. by 1 in., the long axis running parallel with the colon. The anterior lip of the opening was a crescentic band of peritoneum, the posterior lip was not well marked, and was where the parietal peritoneum over the left psoas muscle swept up into the fossa. The sac was about 3 in. deep, and the fundus of it was directed upwards towards the sacrum, and backwards over the brim of the pelvis. The lower or left-hand wall of the sac lay over the left psoas muscle and the left ureter. The upper wall was the root of the pelvic mesocolon and iliac mesocolon. The lining was peritoneum and only slightly thickened. The remainder of the colon and cæcum and mesenteries were normal. Other abdominal viscera were normal. The coronary vessels and aorta were atheromatous.

REVIEWS AND NOTICES OF BOOKS.

The Principles and Practice of Rectal Surgery. By WILLIAM B. GABRIEL, M.S. Lond., F.R.C.S., Surgeon to St. Mark's Hospital and the Royal Northern Hospital. Royal 8vo. Pp. 248 + viii, with 118 illustrations, including 8 coloured plates. 1932. London: H. K. Lewis & Co. Ltd. 20s. net.

THE author says in the preface that the object he had in view when undertaking the task of writing this book was the production of a practical guide to the diagnosis and treatment of diseases of the rectum. We have no doubt that those who read the book will agree that he has succeeded well.

The opening chapter deals with diagnosis, the various methods of examination being minutely described. Excellent advice is given by emphasizing the necessity of re-examination in difficult or obscure cases, and by the warning that a double lesion may exist. The preparation of cases for operation and routine methods of after-treatment are carefully described, much valuable information being given therein. When discussing the various methods of inducing anaesthesia the author shows a decided preference for low spinal anaesthesia by intrathecal stavaine.

The subject of hæmorrhoids is discussed in detail. The method of treatment by injection is fully described and helpful instruction is given as to the efficient manner of carrying it out. Operative treatment is confined to the description of the method of catgut transfixion, which the author prefers to all others. The illustrations of the various steps of the operation might, we think, have been made clearer with advantage.

For the treatment of prolapse of the rectum submucous injections of phenol in oil and perirectal injections of quinine are considered to give the best results.

A novel type of operation is described for the cure of a chronic fissure. A triangular incision is made with a scalpel, starting from the anal margin on each side of the fissure. The lateral incisions vary in length from 2 to 2½ in. and are joined by a transverse incision at their posterior extremity. The length of the transverse incision is not mentioned, but according to the illustration it appears to be about 2 in. long. The skin enclosed by these incisions is removed, with the result that a considerable wound area is left to heal by granulation. It is claimed that the removal of so large an area of healthy skin is necessary to establish drainage for the fissure. The resulting wound takes about five weeks to heal and it is necessary for the patient to desist from running, riding, or playing strenuous games for several months, thus entailing a lengthy convalescence. We fail to see the necessity for sacrificing so large an area of healthy skin, since, in our experience, the simple operation generally performed gives excellent and permanent results, complete cicatrization taking place in three weeks.

For the treatment of ischiorectal abscesses extensive sacrifice of healthy skin is also advocated, subsequent skin-grafting of the granulating surface being necessary to expedite healing.

Seven varieties of anorectal fistulæ are described. Such a classification is purely arbitrary and is not based upon the anatomical sites of the preceding abscess. It is estimated that 15 per cent of anorectal fistulæ are tuberculous.

The etiology, pathology, and the various methods of treatment of pruritus ani are fully discussed. The author has achieved excellent results by the injection of A.B.A. and peraine in oil, his percentage of cures being estimated at over 60 per cent.

The chapter dealing with benign growths is very instructive, the pathological appearances, microscopical as well as macroscopical, being minutely described.

The concluding chapter is devoted to cancer of the rectum, of which a very excellent account is given, including histological classification, methods of routine examination, the value of proctoscopy and sigmoidoscopy, abdominal and general examination, the use of the barium enema, and the interpretation of results. When discussing the spread of cancer of the rectum it is contended that extrarectal spread by means of the lymphatics only takes place after the deep infiltration has passed beyond the rectal wall. It is pointed out that Duke's classification of 215 cancers of the rectum removed by surgical operation shows that in 53 per cent dissemination by means of the lymphatics had not occurred. The natural conclusion, therefore, is that in the 215 operable cases of cancer of the rectum a restricted resection with end-to-end anastomosis would have been sufficient to prevent recurrence in nearly 50 per cent. This seems to us to be an optimistic estimate. It must be extremely difficult to be sure, even after a most thorough examination of a given specimen, that not even a single cancer cell—quite sufficient to give rise to a distant metastasis in the extrarectal lymphatic areas—had escaped detection.

For the surgical treatment of cancer of the rectum the author recommends perineal excision for growths in the ampulla, and perineo-abdominal excision for those at or above the recto-sigmoidal junction. Excellent descriptions of the technique of both operations are given, together with statistical tables setting out the mortality and the survival rates for three and five years.

Little encouragement is offered for treating cancer of the rectum with radium. We agree with the author when he says "a patient's troubles are only just beginning when a radium implantation is given."

We have nothing but praise for this book. It is well written, excellently illustrated, and replete with sound practical instruction.

Chirurgie du Rectum. By HENRI HARTMANN, Professeur de Clinique chirurgicale, Chirurgien de l'Hôtel-Dieu. Imperial 8vo. Pp. 398, with 161 illustrations. 1931. Paris: Masson et Cie. Fr. 75.

This is the best book upon the surgical treatment of diseases of the rectum in the French language with which we are acquainted. It embodies the author's conclusions in regard to the best methods of treating rectal disease, based upon an experience of over thirty years, and is therefore extremely valuable.

The opening chapter is devoted to methods of examination; the various instruments devised for purposes of diagnosis are accurately described and hints given in regard to their proper use. Perirectal suppuration is exhaustively considered in the succeeding chapter, prominence being given to the diffuse forms—namely, septic perirectal cellulitis and diffuse gangrenous phlegmon. It is pointed out, however, that these varieties are not so frequently met with after perineal operations nowadays owing to antiseptic and aseptic methods of procedure. Localized suppuration is considered to be due to septic lymphangitis and phlebitis originating in an abrasion of the mucosa of the anal canal and rectum. Five varieties of circumscribed abscess are described—namely, the submucosa-cutaneous, the submucous, the ischio-rectal, the pelvirectal, and the retro-rectal.

The subject of ano-rectal fistula receives elaborate consideration. The opinion is expressed that, whereas a considerable number of fistulae are preceded by an abscess of septic origin, quite 50 per cent are tuberculous. The usual classification of fistulae into complete, blind external, and blind internal varieties is adhered to, but it is insisted upon that the relationship of the main track to the musculature of the anal outlet is of great importance. Three types of ano-rectal fistulae are described—namely, the subcutaneous, the submucous, and the ischio-rectal. In the author's experience spontaneous cure seldom occurs, and therefore the treatment of the various types of ano-rectal fistulae is by surgical operation. The details of operative procedure are carefully described for each type. An operation which appears to be greatly favoured by the author, the various stages of which are excellently illustrated, is excision of the main track. After completely dissecting out the track as far as the internal opening, the mucosa surrounding the opening is

excised together with the mucosa between the opening and the muco-cutaneous margin. Finally, the rectal mucosa is brought down to the skin margin and sutured there. It is claimed that by excising the internal opening, faecal contamination of the wound is obviated and the wound heals rapidly. It is obvious, however, that the method is only applicable to those fistulæ in which there are no offshoots from the main track.

For the operative treatment of internal hæmorrhoids the author advocates preliminary forcible dilatation of the sphincters. A combined ligature and cautery operation is the method of choice. Whitehead's operation is also recommended.

Prolapse of the rectum receives considerable attention. Only two varieties are recognized—namely, prolapse of the mucous coat only, and total prolapse in which all of the coats of the rectum are involved. For the treatment of total prolapse several surgical procedures are described, including colopecty, posterior rectopecty, removal of an extensive area of mucosa by means of the thermocautery, and amputation of the prolapse by the method advocated by Mikulicz.

The chapter dealing with stricture of the rectum is one of the best in the book. We know of no better account of the etiology of the disease. According to the author's experience, stricture of the rectum occurs almost as frequently in men as in women. In his opinion perineal excision yields the best results, the operative mortality being as low as 5 per cent.

The pathology, symptomatology, and the spread of cancer of the rectum are very lucidly described. Especial stress is laid upon the frequency with which cancer spreads in an upward direction in the pelvic mesocolon along the line of the superior hæmorrhoidal vessels. Kraske's operation is minutely described, and there are excellent illustrations showing the varying amounts of bone removed in the modifications introduced by his followers, notably Hocheneg, Bardenheuer, Rose, and Heinecke. It is stated that the operative mortality of these operations varies from 11 to 20 per cent. Excision by the perineal and the vaginal routes are also well described and illustrated. The operative mortality of perineal excision is estimated at about 14 per cent. The technique of the abdomino-perineal operation is described in detail and is well illustrated. The only point to which we take exception is that the greater part of the peritoneum lining the floor of the pelvis is left behind. In our view the pelvic peritoneum is highly dangerous tissue and should be removed widely. The operative mortality of the abdomino-perineal operation is estimated at 26 per cent, and consequently the author advocates Coffey's two-stage method as a means of reducing the mortality rate.

The concluding chapters are concerned with benign tumours and the operation of colostomy, but these do not call for any special comment.

An Introduction to 'Avertin' Rectal Anæsthesia. By J. KEMPSON MADDOX, M.D., Ch.M. (Sydney), M.R.C.P. (Lond.), Tutor in Medicine, University of Sydney. With a Foreword by HAROLD R. DEW, M.B., F.R.C.S., F.R.A.C.S., Professor of Surgery, University of Sydney. Large 8vo. Pp. 124. Illustrated. 1931. Sydney: Angus & Robertson. 9s. net.

As the author states in his preface, this little book is based on only two years' experience of the use of avertin, and is intended as an interim guide for the assistance of those general practitioners who wish to employ it. In spite, however, of the author's modest description of its aim, it consists of a very able review of the literature on the subject (together with an Anglo-American bibliography) as well as the results of his own experience. No one, whether he be anæsthetist or general practitioner, who has not used avertin could find a more helpful guide than this manual.

The book begins with an historical survey of the use of rectal anæsthesia in general, and the pharmacology of, and general considerations connected with the use of, avertin in particular, the possible complications resulting from its use and their treatment, and finishes with an account of the author's technique, and a general review of the position occupied by the drug in anæsthesia at the time of writing.

The book is easy to read in spite of some ambiguity in parts. The author favours the use of avertin in the majority of cases, especially in the surgery of children and in all those who have repeatedly to undergo painful dressings. Repeated exhibition of the drug in the same patient, as in cases of tetanus, does no harm. He discusses fully its contra-indications and shows how few these are in actual practice.

However, we do not agree with all the statements on the subject, e.g., that it is a perfect relaxing agent in abdominal surgery. If it is so in Australia, the patients there must be rather different from those found in London, or perhaps we should blame the anaesthetics to which the author has been accustomed. His statement that it is the best preliminary medication is open to question, the great drawback to its use in this connection being the technique of its preparation, administration, and posology. In our opinion the newer barbiturates given by the mouth, with or without derivatives of opium and hyoscine, do all that can be done by avertin with only a fraction of the trouble and care necessary in connection with the latter's use. This must not be taken as meaning that there is no place for this drug in anaesthesia, but merely that it is not the only drug that can produce these desirable effects.

In the author's opinion the preliminary cleansing enema can be dispensed with, and in urgent surgery "no harm has resulted from its omission". He also administers the drug only twenty minutes before the operation, though in our experience its full effect is not evident for at least forty-five minutes. He makes an extraordinary statement in the last few pages when he says that either "intravenous avertin" or nitrous oxide is the most suitable anaesthetic agent "for very short operations (e.g., dental extractions)". It is the only reference in the book made to intravenous avertin and one wonders whether he intended to say something else. He also refers to the "initial pain" associated with spinal analgesia, which apparently is peculiar to Australia, as we have no experience of it.

In his treatment of respiratory failure he recommends lobeline " $1\frac{1}{2}$ grains, 0.1 gm." as "an injection", without stating what kind of an injection—whether hypodermic, intravenous, or rectal. In no case have we ever given more than 10 mgrm. at a time, and would hesitate to do so.

On page 80 it is stated that "children up to ten years of age . . . should be given atropin sulphate dissolved in chloroform water by the mouth in a dose of approximately 0.32 mg. ($\frac{1}{100}$ gr.) for each year of age." This statement is obviously made in error, as it would mean, for example, that a child of 10 should be given $\frac{1}{20}$ gr.

Also on page 90 the author states rather ambiguously that "gr. $\frac{1}{6}$ (0.012 gm.) of morphine must be given with 0.11 gm. of avertin per kg. of body weight". His comparative doses are not always correct. On page 90 he writes "gr. $\frac{1}{6}$ (0.012 gm.)" and in another place "gr. $\frac{1}{6}$ (0.010 gm.)", which is the usually accepted equivalent and which he gives in his table at the end of the book.

However, apart from ambiguity here and there and some inconstancy in the relationship between imperial and metric measures and some mistakes in posology, the book is well worth reading. In our opinion avertin is more of a narcotic than either an analgesic or anaesthetic, and this apparently is the opinion of the manufacturers who describe it as a 'basal anaesthetic'.

De l'Exclusion haute de l'Estomac. By BERNARD-OLIVIER GUIHENEUC. Royal 8vo. Pp. 76. 1932. Paris: Imprimerie Lahure.

THIS small pamphlet describes twelve cases in which high exclusion of the stomach was performed for duodenal ulcer (6), pyloric ulcer (2), lesser-curvature ulcer (2), and bleeding ulcers (2). The operation consists of division of the stomach beginning as high as possible on the lesser curvature and reaching the greater curvature at a point slightly to the left of this. On the distal side a simple and complete closure is effected, on the proximal side an anastomosis with the jejunum of 6 to 8 cm. of the stomach at its dependent and divided edge, the rest of the stomach above being closed. At this stage the ulcer is left alone, but at a second operation the middle

and pyloric gastric segments may be removed—in fact, a two-stage partial gastrectomy may be performed.

The point of interest is that the second stage may be dispensed with altogether and the patient suffer in no way, except by the retention of an ulcer scar which may be the seat of malignant disease later. The indications for the operation are chiefly : (1) Hæmorrhage from duodenal and gastric ulcer ; (2) Adhesions round an ulcer making immediate resection difficult ; (3) The serious condition of the patient. The twelve cases are adequately described. One pyloric ulcer and one lesser-curvedure ulcer died from hæmorrhage.

Trattato di Patologia chirurgica generale e speciale. By Prof. OTTORINO UFFREDUZZI (Turin). Large 8vo. Vol. I. Pp. 724 + vi, with 345 illustrations and 8 coloured plates. 1933. Turin : Unione Tipografico-editrice Torinese.

This handsome book, with excellent type and binding, is a credit to the publishers and to the University of Turin. The title, "Surgical Pathology, General and Special", does not convey to English readers quite what it is. The author is known for his practical bent and common sense. He has furnished his students with a text-book of applied pathology that reflects his teaching in wards and theatre, and includes, therefore, more in the realm of treatment than would be found in an English book of similar title.

No fewer than 167 pages are devoted to fractures, not only in their general aspect, but also including details of treatment and prognosis, whilst only 90 pages are spent upon non-traumatic affections of the bones. Similarly 64 pages are given to injuries of joints, and 108 to all inflammatory affections, including tuberculosis.

The present volume also has sections upon the muscles, tendons, bursæ, heart, and vessels. For the general reader the first 150 pages are of greatest interest. There is an historical introduction of 25 pages, which gives Professor Uffreduzzi legitimate opportunity for dwelling upon the greatness of Italy's contribution to the science and art of medicine, but in which he nevertheless presents a fair perspective setting for modern studies. The first section of the text proper deals with repair, the second with inflammation in general, with infection, and with specific infections. As throughout the book, the outlook is sensible and judged from the practical rather than the theoretical standpoint. For example, in dealing with the use of vaccines in acute infections, it is pointed out that stimulation of the organism cannot be lacking when myriads of germs are circulating in the blood, undergoing destruction and pouring endotoxins into the circulation. The subject of tumours in general receives only 16 pages, but the account is very interesting. Incidentally, the views expressed upon irradiation as a means of treatment reflect the Turin School under Bertolotti rather more exclusively than is characteristic of the rest of the book, which constantly reveals the author's acquaintance with the work and literature of Germany, France, America, and England.

A fourth section is headed "Relations between the Theory of the Constitution and Surgical Pathology", and gives opportunity for general talk about heredity, internal secretion, deformities, atavism, anomalies of biochemical activity, and so on. "The individuality factor not only has great value in biology and psychology, but actually dominates the field of pathology. . . ." "For the surgeon, the personality of the supporting tissues, and particularly the connective tissues, is especially important, since it is the essential factor in all reparative processes. . . ." Scores of such sentences could be quoted which are so written as to catch the attention and illuminate a point.

Students who can read this book and remember it will certainly have a grasp of the principles of surgery. It is fully illustrated, and the text figures are adequate, but readers of this JOURNAL will note with some surprise, after reading the list of acknowledgements of Italian sources in the preface, that the best of the plates are extremely familiar to them, and bear the well-known names of Sewell or of Maxwell, where they are accustomed to see them. They may even know the specimens.

Arthritis deformans und chronische Gelenkkrankheiten. By Prof. Dr. HANS BURCKHARDT (Essen). Royal 8vo. Pp. 461 + xii, with 70 illustrations. 1932. Stuttgart: Ferdinand Enke. Paper covers, RM. 53; bound, RM. 55.50.

This monograph, one of the series of 'Modern German Surgery,' contains a thorough and systematic account of chronic specific and non-specific joint diseases. The clinical picture, pathology, treatment, and prognosis of all are well described. The pessimistic view that chronic arthritis follows operations for the removal of the semilunar cartilages with considerable frequency does not agree with the experience of British surgeons; it is interesting to observe that the writer condemns the prevalent Continental practice of dividing the internal lateral ligament in cartilage operations.

For a book of this size the illustrations are few, but most of them have been chosen well. Extensive reference is made to Continental, English, and American literature on the subject.

Die unspezifischen chronischen Erkrankungen der Wirbelsäule. By Prof. Dr. HANS BURCKHARDT (Essen). Royal 8vo. Pp. 77 + vi, with 22 illustrations. 1932. Stuttgart: Ferdinand Enke. RM. 7.50.

This monograph is a continuation of this author's work on chronic joint diseases. It contains an exhaustive account of the chronic non-specific diseases of the spine, some of which are illustrated. Extensive reference is made to Continental literature, chiefly to the work of Schmorl and Calvé.

The Science and Practice of Surgery. By W. H. C. ROMANIS, M.A., M.B., M.Ch. (Cantab.), F.R.C.S. (Eng.), F.R.S. (Edin.), Surgeon and Lecturer on Surgery, St. Thomas's Hospital, etc.; and PHILIP H. MRECHNER, M.D., M.S. (Lond.), F.R.C.S. (Eng.), Hon. Surgeon to H.M. the King, etc. Fourth edition. Royal 8vo. In two volumes. Vol. I, General Surgery. Pp. 865 + x, with 373 illustrations. Vol. II, Regional Surgery. Pp. 1045 + x, with 326 illustrations. 1932. London: J. & A. Churchill. 14s. net per vol.

No better testimony could be given to the value of this work on surgery than the fact that it has within very few years run through four editions. In considering the fourth edition one is naturally particularly interested in those chapters which have been rewritten, or to which new material has been added. That on blood transfusion is sound and practical, but for the education of students it would be well to include the signs and symptoms of imperfect grouping. In the preface it is claimed that the chapter on fractures has been brought into line with modern work, yet space is devoted to Sinclair's skate, and several figures of the ice-tong calipers appear; both of these have surely been superseded by Steinmann's pin, and this has given way to Kirschner's wire. Other revised chapters on the sympathetic nervous system and the spleen are most disappointing; they appear to be written on a classification basis and with a degree of compression quite foreign to the rest of the book. Surely there is sufficient clinical evidence of the value of surgery of the sympathetic system to justify the allotment of more than two pages to this subject?

The authors would appear at times to be lacking in a sense of clinical balance; thus six different methods of performing partial gastrectomy are spoken of, but the treatment of acute intestinal obstruction is dismissed in less than a page, and surely it is in the province of a text-book to discuss treatment rather than operative detail? As additional space has been required for chapters on new subjects, some of it could have been spared in the chapter on amputations (which still contains much that is obsolete), even if operations of historic interest, which one scarcely expects to be included in text-books, were not taken into account. It is hoped that in the next edition the authors will use the blue pencil unsparingly both in the elimination of letterpress and of figures of subjects of no clinical importance, such as those of mammary hypertrophy or one of malignant pustule, which would not suggest the disease in question to anyone of wide acquaintance with it.

In spite of these defects, which are possibly those of any text-book passing rapidly through several editions, the work aims at and attains a high level, which is probably unsurpassed by that of any other modern text-book in the English language. Nevertheless, the authors, by taking their courage in their hands and excluding any prolonged description of conditions which they had never seen in their own hospital for the last ten years, would have found that they had saved sufficient space to devote more pages to subjects of increasing clinical interest, and probably also to reduce the two volumes appreciably in size.

Idols and Invalids. By JAMES KEMBLE, Ch.M., F.R.C.S. Crown 8vo. Pp. 212 + x. 1933. London: Methuen & Co. Ltd. 6s. net.

MR. KEMBLE has collected together a series of essays, some of which have already appeared in various magazines. His object is to explain on medical grounds the conditions which have modified the lives and actions of some persons known to history. The results of his inquiries are often interesting, although they will not always be accepted by his fellow-historians. The most satisfactory essay perhaps is that dealing with Judge Jeffreys, who was born in 1648 with so brilliant a brain that he became Lord Chief Justice at the age of 35. Two years later his character changed rapidly for the worse, and in 1685 he presided at the Bloody Assizes which have handed his name down in infamy. Mr. Kemble quotes a letter written from Dorechester during the Western Circuit, in which he says, "I am so tortured with the stone as to have to make use of my servant's pen to give a relation of what has happened since I came here." The pain of the vesical calculus, coupled with an acute cystitis increased by long journeys over the rough roads of the West Country, was, Mr. Kemble thinks, a sufficient explanation of the outbreaks of temper and the monstrous judicial interruptions which marked the trials at Salisbury, Exeter, Taunton, Bristol, and Wells. The explanation is plausible and probably correct.

The essays on Columbus and King Henry VIII are more open to criticism. Mr. Kemble thinks that the mental symptoms from which Columbus is known to have suffered late in life were associated with G.P.I. He accepts without demur the general belief that Henry VIII was syphilitic, but this essay seems to have been written before the publication of Mr. Fredrick Chamberlin's painstaking study of the private character of Henry VIII.

There are interesting accounts of Byron, of Louis XV, of the Borgias, of Cleopatra, of Nelson, and of Queen Anne. The book ends with some extracts from Ramazzini's *Treatise on Diseases of Tradesmen* translated into English in 1746. It is an early treatise on occupational diseases.

Urology in Women. A Handbook of Urinary Diseases in the Female Sex. By E. CATHERINE LEWIS, M.S. (Lond.), F.R.C.S., Surgeon, Royal Free Hospital, London. Large 8vo. Pp. 76 + viii, with 21 plates and 4 illustrations in the text. 1932. London: Baillière, Tindall & Cox. 6s. net.

THE authoress states in the preface that this little book is not intended to be a complete treatise on urology, but rather to emphasize certain points which have a particular application in women patients. She has succeeded admirably, for the book is pleasantly written, well printed and illustrated, light enough to read in an easy chair, and published for the unusually low price of six shillings. We are sure that there is a definite opening for such monographs, if only for reading on a railway journey.

There are some faults in the book, owing perhaps to the authoress's commendable efforts to make it readable: thus on page 27 she writes of repeating an examination on a *sterile* specimen of urine when she almost certainly means one that has not been contaminated; and when she refers to a growth of the urethra as being "either comparatively innocent or suspiciously rank in appearance", she makes use of a description which is likely to cause acute pain to a sensitive mind. In the article

Over thirty countries took part in the Congress, and the average English professional man finds great difficulty in following the discussions in such a wide diversity of languages; hence the value of this report, which in a small volume of 166 pages embodies most that is important in the debates which in the original official report occupy 763 pages.

We have heard it said that these International Congresses resemble the building of the tower of Babel with a confusion of tongues and no conclusion.

This, however, only refers to the papers and debates, and there is no doubt that the military medical services of all countries have derived much knowledge of the methods adopted by other countries to deal with sick and wounded in war since these bi-annual congresses were inaugurated. But of even greater value is the personal contact with the leaders in military medical matters of all the countries concerned.

The only weak point in Commander Bainbridge's report is the lack of reference to the various demonstrations which are always a feature of these Congresses.

The exhibition of Field Medical Units, instruments and appliances, and visits to military hospitals and establishments give the visitor a better idea of the methods adopted in a particular country than the papers read and the discussions which follow.

At this Congress, held in 1931 at The Hague, five subjects were put up for discussion.

- I. The recruiting, training, and advanced training of military medical officers and pharmacists.
- II. The psychoneuroses of war: the immediate and remote effect of war on the nervous system of combatants and non-combatants.
- III. Methods of haemostasis on the battlefield: standardization of first aid material and the mode of application.
- IV. The preparation and storage of medicinal ampoules in use in the naval and military medical services.
- V. The sequele of war wounds of the teeth and inferior maxilla: their treatment.

In this country we are all aware of the difficulty experienced since the great war in attracting suitable and sufficient young medical officers to the fighting services. These difficulties would appear to be just as great in other countries.

While some of the nations advocated the taking over of the complete medical education of students designed for the Services, from the outset of their career, so that the importance of military problems would be constantly kept in view, the majority were agreed that it is better for the officer to obtain a civil qualification first and then undergo a period of special training in the application of his knowledge to service conditions in peace and war.

The latter method, which is adopted in this country, is undoubtedly the correct one.

All were agreed that the low emoluments of military surgeons were the great bar to getting candidates.

In this country we have no corps of pharmacists such as exists in most continental armies.

These pharmacists form a highly trained body of commissioned officers who are not only responsible for all questions of medical and surgical supply but who also undertake analysis, supply sera, and carry out disinfection.

In the British Army much that is done by the pharmacist is the province of our pathological and hygiene departments.

While we are never likely to have a similar body in our army the possibility of having a few commissioned pharmacists as advisors and as instructors of our non-commissioned officer dispensers might warrant consideration.

The wish, expressed in the conclusions of the Congress on this subject, that exchange between the medical officers of the various countries should take place is a sound one, but unlikely to be fulfilled until the serious shortage of regular officers is adjusted.

Nothing very new emerged from the discussion on the second subject, and all the points brought out in the conclusions have already been in operation in this country.

Subject three is of more direct surgical interest. All contributors to this discussion laid great emphasis on the dangers of the elastic tourniquet. The result of the conclusions on this matter have borne fruit, and modifications in the training of stretcher bearers and medical personnel have been adopted.

Subject four was of special interest to the pharmacists. In our army we rely largely on the trade for medicinal products put up in ampoules.

The fifth subject provided an interesting and important discussion, and although few new points were raised, all speakers stressed the necessity of team work between surgeon and dentist when dealing with facio-maxillary injuries and the establishment of definite centres both in the fighting area and at home for the treatment of such cases.

This was recognized by this country early in the great war, and all the recommendations put forward have been adopted already by us—in fact the conclusions are based largely on our system.

The report has a short foreword from the Surgeon-General, U.S. Army.

It can be recommended as a correct and useful account of the proceedings of the Congress, and should find a place in the library of all those who are interested in military medical and surgical problems.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Modern Aspects of Gastro-enterology. By M. A. ARABA, M.R.C.P. (Lond.). Medical Assistant to Guy's Hospital, etc. With a Foreword by ARTHUR F. HURST, M.D., F.R.C.P., Senior Physician, Guy's Hospital. Large 8vo. Pp. 374 — xviii, with 79 illustrations. 1933. London: Baillière, Tindall & Cox. 27s. 6d. net.

A Synopsis of Surgery. By ERNEST W. HEY GROVES, M.S., M.D., B.Sc. (Lond.). F.R.C.S., Consulting Surgeon to the Bristol General Hospital, etc. Tenth edition. Crown 8vo. Pp. 693 — viii, with 163 illustrations. 1933. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

Peripheral Nerve Injuries. By LEWIS J. POLLOCK, M.D., Professor of Nervous and Mental Diseases, North-western University, etc.; and LOYAL DAVIS, M.D., Professor of Surgery, North-western University, etc. Imperial 8vo. Pp. 678 — xx, with 312 illustrations. 1933. New York: Paul B. Hoeber Inc. \$10.00.

Intracranial Tumours Roentgenologically Considered. By LOYAL DAVIS, M.D., Ph.D., F.A.C.S., Professor of Surgery, North-western Medical School. Vol. XIV of Annals of Roentgenology, edited by JAMES T. CASE, M.D., Professor of Roentgenology, North-western University. Large post 4to. Pp. 277 — xviii, with 135 illustrations. 1933. New York: Paul B. Hoeber Inc. \$10.00.

Die kombinierte Enzephal-Arteriographie. By Prof. Dr. W. LÖHR and Prof. Dr. W. JACONI (Magdeburg). Imperial 8vo. Pp. 83, with 75 illustrations. 1933. Leipzig: Georg Thieme. Paper covers, M. 16; bound, M. 18.

Der Bandschaden des Kniegelenks. By Dr. KARL GERHART (Munich). Medium 8vo. Pp. 59, with 21 illustrations. 1933. Leipzig: Johann Ambrosius Barth. RM. 4.50.

Surgery of the Thorax. By T. HOLMES SELLORS, M.Ch., M.A., B.M. (Oxon.), F.R.C.S., Assistant Surgeon to Queen Mary's Hospital for the East End, etc. With a Preface by R. A. Young, C.B.E., M.D., F.R.C.P. Demy 8vo. Pp. 519 — xxv, with 140 illustrations, including 11 plates in colour. 1933. London: Constable & Co. Ltd. 22s. 6d. net.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, etc. Fourth edition, revised and enlarged. Large 8vo. Pp. 287 — xx, with 335 illustrations, some of which are in colour. 1933. Bristol: John Wright & Sons Ltd. 21s. net.

- Vorbeugung und Bekämpfung der Operationsgefahren.** By Prof. Dr. M. KAPFIS (Hannover). Royal 8vo. Pp. 382 + xii, with 12 illustrations. 1933. Leipzig: Georg Thieme. Paper covers, M. 17; bound, M. 18.60.
- Radium: Production, General Properties, Therapeutic Applications, Apparatus.** Demy 8vo. Pp. 346, illustrated. 1933. London: Chemical Service Co. Ltd., Sunic House, Kingsway, W.C.2.
- Die Bluttransfusion in Theorie und Praxis.** By Dr. Med. HANS WILDEGANS, A. O. Professor für Chirurgie an der Universität Berlin. Medium 8vo. Pp. 157 + iv, with 36 illustrations. 1933. Berlin: Julius Springer. RM. 9.60.
- Précis d'Anatomie pathologique.** By GUSTAVE ROUSSY, Professeur à la Faculté de Médecine de Paris; ROGIER LEMOIX and CHARLES OBERLING, Professeurs agrégés à la Faculté de Médecine de Paris. Large post 8vo. Two volumes. Pp. 1344, with 582 illustrations and 4 coloured plates. 1933. Paris: Masson et Cie. Paper covers, Fr. 160; bound, Fr. 175.
- The Diagnosis and Treatment of Postural Defects.** By WINSTROP MORGAN PHELPS, B.S., M.D., M.A., F.A.C.S., Professor of Orthopaedic Surgery, Yale University, etc.; and ROBERT J. H. KIMMUTH, Assistant Professor of Physical Education, Yale University. Royal 8vo. Pp. 180 + xiv, with 108 illustrations. 1933. London: Baillière, Tindall & Cox. 23s. net.
- Papers Relating to the Pituitary Body, Hypothalamus, and Parasympathetic Nervous System.** By HARVEY CUSHING, Professor of Surgery (Emeritus), Harvard University, etc. Large 8vo. Pp. 231 + viii, with 99 illustrations. 1933. London: Baillière, Tindall & Cox. 29s. net.
- Roentgenographic Studies of the Urinary System.** By WILLIAM E. LOWIE, M.D., F.A.C.S., Chief of Department of Urology, Cleveland Clinic, etc.; and BERNARD H. NICHOLS, M.D., F.A.C.R., Chief of Department of Roentgenology, Cleveland Clinic. Imperial 8vo. Pp. 812 + xviii, with 812 illustrations. 1933. London: Henry Kimpton. 84s. net.
- Atlante di Chirurgia dello Stomaco.** By G. EGIDI. Large 8vo. Pp. 15, with 30 plates. 1933. Rome: Luigi Pozzi. L. 35.
- The Cure of Hæmorrhoids, Varicose Veins and Ulceration, and Allied Conditions: by Modern Methods and Bandaging.** By STUART McAUSLAND, B.A. (Lond.), M.D., Ch.B. (Liv.), M.R.C.S., L.R.C.P., Hon. Physician, Hahnemann Hospital, Liverpool, etc. Large 8vo. Pp. 63. Illustrated. 1933. London: John Bale, Sons and Danielsson. 3s. 6d. net.
- Operative Surgery.** By WARREN STONE BICKHAM, M.D. and PHAR. M. (Tulane), M.D. (Columbia), F.A.C.S., Former Surgeon in charge of General Surgery, Manhattan State Hospital, New York, etc.; and CALVIN MAXON SMYTH, JR., B.S., M.D., F.A.C.S., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania, etc. Vol. VII, The Newest Operations and General Index. Royal 8vo. Pp. 849 + viii, with 765 illustrations. 1933. Philadelphia and London: W. B. Saunders Co. 55s. net.
- Die Bruchoperation nach Bassini.** By Dr. ATTILIO CATTARINA, O.-O. Professor der chirurgischen Operationslehre an der kgl. Universität in Genua. Large crown fo. Pp. 56, with 16 coloured plates by HONORIUS GAVIGNI, Doktor der gesamten Heilkunde. 1933. Berlin and Vienna: Urban und Schwarzenberg. RM. 12.
- Hirnehirnchirurgie: Erfahrungen und Resultate.** Edited by Dr. HANS HOFF and Dr. LEOPOLD SCHÖNBAUER (Vienna). Royal 8vo. Pp. 472 + vi, with 238 illustrations. 1933. Leipzig and Vienna: Franz Deuticke. Paper covers, M. 40; bound, M. 43.
- The Injured Workman.** By G. F. WALKER, M.D., M.R.C.P., with the collaboration of J. HARVEY ROBSON, Barrister-at-Law (Law); R. E. JOWETT, M.D., Ch.B., D.L.O. (Ear, Nose, and Throat Surgery); STANLEY RITSON, M.B., M.S., B.Sc., F.R.C.S. (General Surgery); JOHN FOSTER, M.A., M.B., F.R.C.S., D.O.M.S. (Ophthalmic Surgery). Foreword by W. H. MAXWELL TELLING, M.D., B.S., F.R.C.P., Professor of Forensic Medicine, University of Leeds. Crown 8vo. Pp. 190 + xx. 1933. Bristol: John Wright & Sons Ltd. 6s. net.
- The Digestive Tract. A Radiological Study of its Anatomy, Physiology, and Pathology.** By ALFRED E. BARCLAY, O.B.E., M.A., M.D., D.M.R.E., Lecturer in Medical Radiology, University of Cambridge. Large 8vo. Pp. 395 + xxviii, with 275 illustrations. 1933. Cambridge: University Press. 36s. net.

THE BRITISH JOURNAL OF SURGERY

VOL. XXI.

OCTOBER, 1933.

No. 82.

THE CLINICAL ASPECTS OF BRANCHIAL FISTULÆ.

By HAMILTON BAILEY,

SURGEON, ROYAL NORTHERN HOSPITAL, LONDON.

In a fœtus approximately thirty days old at least four well-developed grooves can be seen on the side of the neck. Commonly these are termed 'branchial clefts' (*Fig. 105*), and the intervening bars 'branchial arches'. In human embryos these clefts are, correctly speaking, grooves — grooves on the outside, and on the pharyngeal aspect separated by a layer of mesoblast. The clefts begin to appear about the tenth day, when the embryo is about 2.6 mm. in length. Forty days later, or even in less time, the second, third, and fourth clefts have disappeared. There can be little wonder that in studying so transitory and minute a structure as the branchial apparatus in material which is difficult to obtain, differences of opinion have arisen concerning the origin of branchial fistulæ.

ORIGIN OF BRANCHIAL FISTULÆ.

Rabl's Theory. — The one basic fact to which all embryologists subscribe is that

the Eustachian tube is the persistent first cleft, and that the tympanic membrane represents the septum between the external and the internal grooves. Rabl¹ homologized a complete branchial fistula with the auditory canal, suggesting that the septum (tympanic membrane) had perforated early



FIG. 105.—Fœtus showing branchial grooves and arches.

in embryonic life. From a study of anatomical relationships Rahl agreed with other observers that the cleft usually implicated was the second. This has been termed the 'arm-chair' theory. Until it has been definitely disproved its simplicity is bound to make an appeal to the clinical worker. It is a seductive theory; one merely has to assume that the branchial grooves are the atavistic remnants of the fish's gills, and lo! the patient with a branchial fistula before us becomes one more example illustrating Crile's² conception of man as a giant amoeba slowly creeping up the slippery sides of time.

Many of those who are best able to judge³⁻⁵ deride this theory. J. E. Frazer,³ whose works on embryology are so well known, objects to the very word 'branchial'. He states that branchial fistulae have nothing to do with the branchiae, and in many cases are outside the region which might be homologized with the gills of lower vertebrates.

Wenglowski's Theory.—This is the very antithesis of an 'arm-chair' theory. This patient Russian¹ worked for five years on the branchial apparatus, examining no fewer than 75 embryos and 246 cadavers before propounding his theory in 1912. An opinion based upon premises such as these justly commands respect. He presented evidence to show that the branchial apparatus cannot leave remnants in the neck below the level of the hyoid. His conclusion is that a branchial fistula, if the term may be allowed, is a persistent thymic duct, and that this thymic duct originates from the third pharyngeal pouch, whence it pursues a long oblique course down the neck, to reach the upper part of the back of the sternum.

Even if I were qualified to do so, it is not my desire to become involved in the intricacies of the embryology of this difficult region. My object has been to approach the subject from the purely clinical side, but I have fostered a hope that by dropping a morsel of truth from the patient's neck into the seething cauldron of controversial cervical embryology, perchance I might help to clarify what is to be served to the student to-morrow.

CLINICAL FEATURES.

Unlike a thyroglossal⁷ fistula, a branchial fistula is usually present from birth. While in none of my cases has there been any hereditary factor, there are cases on record where the abnormality has been transmitted,⁸⁻¹⁰ usually through the mother. Females appear to be more often affected in a proportion of 3 : 1. When the fistula is unilateral the right side is said to be more often affected. It happens that in the five unilateral cases under my observation all have been right-sided.

Branchial fistulae are of three distinct types : (1) Complete, with an outer and an inner opening ; (2) Incomplete external fistula, with an outer opening only ; (3) Incomplete internal fistula, with an inner opening only. The commonest variety is blind internally, the rarest blind externally.

The External Opening.—In fully 80 per cent of those cases which have an external orifice that orifice is situated in the lower third of the neck, opposite the anterior border of the sternomastoid (*Figs. 106, 112*). In this connection it is interesting to compare and contrast a typical branchial cyst^{11, 12} with a typical branchial fistula.



FIG. 106.—A branchial fistula of over thirty years' duration. *Inset*: X-ray after injecting the fistula with lipiodol.



FIG. 107. —Cervical aneurism. These appendages are believed to have been common in the days of the Romans, for Sir John Bland-Sutton has drawn attention to examples of statuary of this period where large bilateral cervical aneurisms are depicted by the sculptor proof, indeed, that they were common enough to attract notice.

FISTULA

1. Present at birth.
2. Situated in the lower third of the neck.
3. Lined with columnar epithelium, usually ciliated.
4. Exudes sticky mucus.



Very occasionally the orifice has been observed at the posterior border of that muscle; in a figure by Semken¹⁴ the orifice is shown in the posterior

FIG. 109.—Section of the branchial cartilage in Fig. 108.

triangle of the neck. In assessing atypical external apertures we must inquire into the possibility of the fistula being acquired, an example of which is afforded by the following case:—

CYST

- Appears in early adult life.
- Situated in the upper third of the neck.
- Lined with stratified squamous epithelium.
- Filled with opaque fluid rich in cholesterol.

The lower third of the neck opposite the anterior border of the sternomastoid is also the situation *par excellence* of two other rare branchial abnormalities—cervical auricle (Fig. 107) and persistent branchial cartilage¹³

FIG. 108.—A persistent branchial cartilage. It lay in the subcutaneous tissue tethered to a dimple in the skin situated where a branchial fistula commonly opens. By stretching the skin, as was done while this photograph was taken, the cartilage became more apparent.

(Figs. 108, 109). These structures can be homologized clearly with the pinna.

Atypical External Openings.—In a small proportion of cases the external orifice is situated midway along the anterior border of the sternomastoid.



Mrs. G. C., aged 35, was sent complaining of a fistula which discharged mucus and pus (*Fig. 110*). Twenty years previously a swelling in the neck had been incised, and the resulting sinus continued to discharge. At varying intervals the

FIG. 110.—An acquired branchial fistula of over twenty years' duration. A branchial cyst had been incised during one of the periodic attacks of inflammation to which these cysts are subject.

discharge ceased, and this was usually followed by subacute inflammation on the right side of the neck. The indurated fistula, which extended up to the base of the skull, was dissected out in the manner presently to be described. The result was entirely satisfactory.



An Internal Orifice.

—For a fistula to have an internal orifice is distinctly uncommon. Such an opening is usually situated in the region of the tonsil,¹⁵ most often in the fossa of Rosenmüller (*Fig. 111*). The relation of the tonsil to the innermost limit of a patent or potential branchial

FIG. 111.—Complete branchial fistula extending from the lower third of the neck to the posterior tonsillar recess.

fistula is important. Kramer¹⁶ reported a case of an internal cervical fistula which followed tonsillectomy. I have seen a branchial cyst appearing for the first time a fortnight after tonsillectomy, and Johnson¹⁷ reported a similar case.

SYMPTOMS.

Apart from the continual or intermittent discharge of mucus, which is somewhat annoying, the leading symptoms are due to recurrent attacks of inflammation in the fistula. In cases with an external orifice these inflammatory attacks are fairly constant, and often it is during one of these attacks that the patient seeks advice (*Fig. 112*).

The orifice of the fistula is commonly small and relatively inconspicuous, although a few cases have been described when a complete fistula was so large



FIG. 112.—Bilateral branchial fistulae; left side inflamed.

that the patient's complaint was that bread crumbs and other particles of food passed through it.¹⁸

Sometimes the vagus is a direct relation of the fistula, and probing, pinching, or injecting these fistulae has produced irritative vagal symptoms such as cough, palpitation, and an intermittent pulse. Carp¹⁹ described the case of a boy of 5 with a branchial fistula who gave a history of an unproductive cough of two years' duration unsuccessfully treated by tonsillectomy and medical measures. At operation the tract was found adherent to the vagus, and excision of the fistula cured the cough. Similar examples have been reported.

The externally blind variety appears to be rare. It is possible that they

are more common than is generally supposed, and that a fine-calibred tract discharges mucus unobtrusively into the fossa of Rosenmüller. Douglas²⁰ reports a case of a man who, when he closed his mouth and occluded his nostrils, could force air into his blind fistula and distend his neck. When this variety of fistula is complicated by having a large lumen it is liable to become a source of danger. Eddowes's patient was a nurse of 30 whose fistula became periodically full of purulent material, and absorption of toxins from the pus sac undermined the patient's health.

INVESTIGATION AND TREATMENT.

If the tract shows evidence of recent inflammation it should be treated conservatively for at least fourteen days.

To ascertain the extent of the fistula there is no better method than a lipiodol injection. The following method of investigation and treatment has several notable advantages. Three or four hours before the proposed operation for complete extirpation of the fistula the skin about the orifice is infiltrated with local anæsthesia, and a purse-string is introduced *very close to the periphery of the opening*. At this stage the suture is not tied. If the external orifice will admit a ureteric catheter, this is introduced. More often it will be found that the opening is too small, when a lachrymal-duet syringe can be employed (*Fig. 113*). Lipiodol does not run freely through a fine-nozzled syringe, but diluted with an equal volume of liquid paraffin and well stirred it will be found to flow tolerably, while the diluted medium's opacity to X rays is in this instance sufficient to cast a good shadow. During the injection due regard must be given to possible reflex vagal symptoms; I have perhaps been fortunate not to have observed any disturbances from this cause. When it is judged that the sinus is distended with the medium the syringe is removed while the purse-string suture is drawn taut and tied. The patient can now be sent to the radiological department and X-rayed without the annoyance of the lipiodol escaping, which only too often occurred when employing other techniques. The pictures obtained by the method are instructive (*see Fig. 106. Inset*).

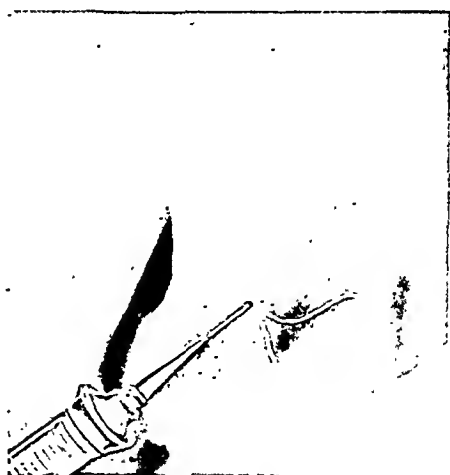


FIG. 113.—Method of injecting a cervical fistula with lipiodol. A subcutaneous purse-string suture has been inserted and a lachrymal-duet syringe is being employed.

Complete Extirpation of a Branchial Fistula.—The neck and the purse-string stitch have been kept sterile during the time (usually a few hours) which intervenes between the X-ray and the operation. An oblique, mainly transverse, incision is made about the external orifice, taking care that the

purse-string suture is not cut in the process (*Fig. 114*). Once the skin is freed about the orifice of the fistula, the free ends of the purse-string can be caught in a hæmostat, and in this manner gentle traction can be applied to the fistula. Instead of the tract becoming more and more difficult to dissect, it will be found usually that sinuses distended with lipiodol, which converts them into elongated cysts, can be freed from surrounding structures with comparative ease. So the dissection proceeds until mild traction upon the fistula and reasonable retraction of the upper limit of the wound fails to give the most perfect exposure.

The time has now come when a second incision should be made in the skin parallel with the first, but at a higher level (*Fig. 115*).



FIG. 114.—The step-ladder method of excising a branchial fistula. Stage 1.

The tract is threaded through from the first to the second incision and the dissection is begun anew. This 'step-ladder' method leaves quite inconspicuous scars (*Fig. 116*), and robs the dissection of a branchial fistula of most of its terrors. The last part of the dissection is often rather difficult. I have noted on two occasions that the tract ran between the external and the internal carotid arteries to reach the pharyngeal wall. On another occasion the tract, which had been the seat of many attacks of inflammation, was intimately adherent to the jugular vein just below its bulb.

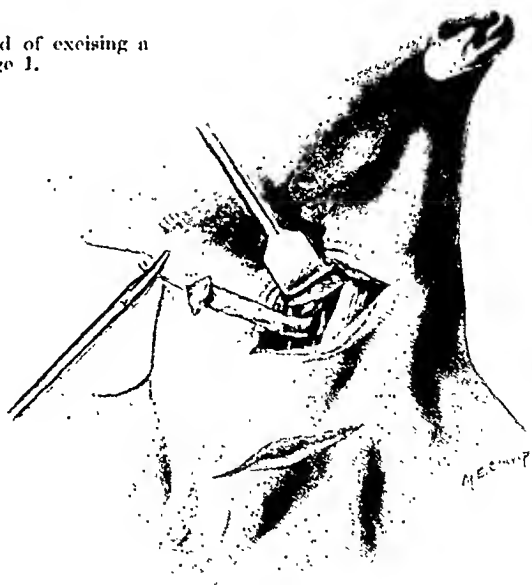


FIG. 115.—The step-ladder method of excising a branchial fistula. Stage 2.

Several operators have had to make a special dissection of the vagus from the wall of the fistula, which, if it is long enough, always appears to dive towards the pharynx beneath the posterior belly of the digastric. It behoves us to have adequate exposure, particularly during the last stages of the dissection, and the dual incision gives us just that exposure we need without leaving the patient, often a young woman, with a deforming scar.

The dissection completed, there remains only to close the skin according to individual preference. It is usually advisable to provide drainage through a stab-puncture wound below the first incision—that is, just above the clavicle. By employing the dual incision the less satisfactory and probably more dangerous methods of inverting the pharyngeal end^{5, 21} of the fistula become unnecessary and need not be described. With proper exposure every fistula can be cleanly dissected from the neck. In bilateral cases it is distinctly unwise to attempt anything more than the extirpa-



FIG. 116.—Patient six weeks after excision of a branchial fistula by the step-ladder method.

tion of one fistula at a sitting.



FIG. 117.—Typical section of a branchial fistula, showing the ciliated columnar epithelium.

Histology of the Excised Tract.—Typically a branchial fistula is lined by columnar ciliated epithelium (*Fig. 117*). Such a lining is practically a constant finding unless there have been many recurrent attacks of inflammation in the fistula, in which case the epithelial lining may be partially or totally denuded. In a number of cases muscle fibres are present in the walls of the tract. As in the case of branchial cysts, there is often an ensheathing layer of lymphadenoid tissue.

CONCLUSIONS ON THE DERIVATION OF BRANCHIAL FISTULÆ FROM A CLINICAL STUDY.

1. Much as we may admire Wenglowski's assiduous labour, his statement that the branchial apparatus never leaves remnants in the neck below the level of the hyoid—a statement which is the keynote of his hypothesis—must be incorrect. *Figs. 108, 109* illustrate a persistent branchial cartilage—a remnant, the origin of which cannot be disputed—situated in the lower third of a child's neck in the exact position where a branchial fistula commonly opens.

2. A cervical auricle, by its structure clearly a homologue of the pinna, is situated at the point where a branchial fistula usually opens.

3. Histologically a branchial fistula is usually lined by columnar epithelium, and its walls sometimes contain muscle. There is no reason why a thymic bud should contain these elements.

4. The anatomical relationships of a branchial fistula as displayed during an operation for its extirpation are in keeping with those depicted in works on embryology as belonging to the cervical sinus and the second cleft which persists in the goat to open into the fossa of Rosenmüller.

REFERENCES.

- ¹ RAHL, C., *Prag. med. Woch.*, 1886, xi, 497; 1887, xii, 3.
- ² CHILL, G. W., *Surg. Gynecol. and Obst.*, 1924, xxxviii, 431.
- ³ FRAZER, J. E., and BERTWISTLE, A. P., *Brit. Jour. Surg.*, 1924-5, xii, 561.
- ⁴ WENGLOWSKI, R., *Arch. f. klin. Chir.*, 1913, c, 789.
- ⁵ MEYER, H. W., *Ann. of Surg.*, 1932, xcv, 1, 226.
- ⁶ CHRISTOPHER, F., *Surg. Gynecol. and Obst.*, 1924, xxxviii, 329.
- ⁷ BAILEY, HAMILTON, *Brit. Jour. Surg.*, 1924-5, xii, 579.
- ⁸ VAUGHAN, A. E., *Brit. Med. Jour.*, 1899, i, 148.
- ⁹ HYNDHAM, O. R. and LIGHT, G., *Arch. of Surg.*, 1929, xix, 410.
- ¹⁰ EDDOWES, A., *Brit. Med. Jour.*, 1920, i, 326.
- ¹¹ BAILEY, HAMILTON, *Brit. Jour. Surg.*, 1922-3, x, 565.
- ¹² BAILEY, HAMILTON, *Clinical Jour.*, 1927, lvi, 619.
- ¹³ BAILEY, HAMILTON, *Ibid.*, lx, 409.
- ¹⁴ SEMKEN, G. H., *Nelson's Loose Leaf Surgery*, ii, 783.
- ¹⁵ SHEDDEN, W. M., *New Eng. Jour. of Med.*, 1931, ccv, 800.
- ¹⁶ KRAMER, R., *Laryngoscope*, 1926, xxxvi, 517.
- ¹⁷ JOHNSON, J. A., *Minnesota Med.*, 1926, ix, 514.
- ¹⁸ FISHER, quoted by Leegaard, *Arch. f. Laryngol.*, 1912, xxvi, 125.
- ¹⁹ CARP, L., *Surg. Gynecol. and Obst.*, 1926, xlii, 772.
- ²⁰ DOUGLAS, J., *Ann. of Surg.*, 1918, lxxvii, 240.
- ²¹ LILIENTHAL, H., *Ibid.*, 1922, lxxv, 119.

THE DIVERTICULA OF THE JEJUNO-ILEUM.

By IAN FRASER,

ASSISTANT SURGEON, BELFAST HOSPITAL FOR SICK CHILDREN.

“The Diverticula and blind bypaths in
Which sophistry and deceit are wont to tread.”

HALES—*Golden Remains*.

IN the Century and Oxford Dictionaries a diverticulum is described as a ‘way-side shelter or lodging’, with, from the context, the underlying meaning that they are houses of ill repute where trouble is apt to brew. In the large intestine they live up to their bad reputation, and as a temporary lodging for bowel contents can give rise to endless trouble. As a contrast the small intestine is interesting, for the condition can be present to an advanced degree and yet the patient be free from suggestive symptoms. Such probably is natural, when one considers the rapidity of the intestinal stream and fluidity of the contents in the small as compared with the large intestine. The difference in the anatomical features of diverticula in the two regions is probably also a factor (*Table I*).

Table I.—COMPARISON OF FALSE DIVERTICULA IN LARGE AND SMALL INTESTINE.

LARGE INTESTINE	SMALL INTESTINE
<i>Sex.</i> —Male preponderance 4-1.	.. Male preponderance 3-1.
<i>Age.</i> —Average 60.	.. Average 60.
<i>Causation.</i> —Spastic colon.	.. Increased intra-intestinal pressure.
Definitely related to situation of artery.	.. Definitely related to situation of artery.
<i>Symptoms.</i> —Left iliac fossa : discomfort.	.. Vague indigestion. Flatulence, borborygmi.
Constipation, flatulence.	Occult blood. May be symptomless.
May be tumour. May be symptomless.	
<i>Complications.</i> —Perforation with local or general infection.	.. Perforation, local abscess or general peritonitis. Adhesions, kinking and obstruction.
Adhesions, kinking, obstruction, fistula formation.	
<i>Region.</i> —Majority sigmoid colon, 70 per cent.	.. All start in upper jejunum.
<i>Situation.</i> —Between the longitudinal bands.	.. Mesenteric border.
<i>Spread.</i> —Into epiploic bodies and leaves of mesentery.	.. Into leaves of mesentery.
<i>Size.</i> —Cherry stone to cherry.	.. Cherry to orange.
<i>Shape.</i> —Retort-shaped.	.. Spherical or mushroom.
<i>Neck.</i> —Narrow in proportion—closed—firm margin.	.. Large in proportion—open.
<i>Contents.</i> —Inspissated faeces or mæcin.	.. Fluids, gas, or empty.
<i>Induration.</i> —Usually present.	.. Nil.
<i>X Ray.</i> —Usually positive and definite.	.. Often negative. When positive conclusive.
<i>Malignancy.</i> —Possible forerunner.	.. No connection.
<i>Diverticulitis.</i> —Common.	.. Rare.
<i>Perforation.</i> —Common.	.. Rare.

It is not here intended to deal with all the diverticula found in the small intestine, but rather with one type only—the multiple false variety.

CLASSIFICATION OF JEJUNO-ILEAL DIVERTICULA.

Of the diverticula occurring in the jejunum-ileum there is no satisfactory classification. The writer would tentatively suggest the following as a basis from which to work, and one which, with greater knowledge, might be further subdivided: (1) Multiple false diverticula; (2) Meckel's diverticula; (3) Congenital enterogenous diverticula; (4) Anomalous diverticula.

Anomalous Diverticula.—Taking these in the reverse order, one would include under the last group those irregular sacs which occur along the intestine due to (a) the drag of a tumour, (b) the pull of adhesions, (c) the softening resulting from caseous glands or tuberculomata. Naturally such pouches may vary in size, shape, position, and appearance, depending upon the causative force. No clinical syndrome can be drawn up for them. They may not necessarily recur in individuals true to type, and are best included under the general term 'anomalous'.

Enterogenous Diverticula.—This group of diverticula has been dealt with in this JOURNAL very fully in an excellent article by Arthur Evans. Suffice it to say that these sacs arise primarily as isolated masses of cells, which separate off as small islands from the primitive gut. These islands by becoming vacuolated in the centre form cysts, and if later a secondary attachment is made to the intestine, they produce diverticula. Their interest lies in the fact that they are composed of a wall similar to the gut, i.e., muscle, mucous membrane, goblet cells, etc. They can occur at almost any point along the course of the small intestine attached to the mesenteric or anti-mesenteric border. Their origin helps to explain most of those isolated unique diverticula reported in the literature from time to time. In shape and appearance they may vary a great deal, yet a common etiology and pathology groups them together and allows them all to be included in the same subdivision.

Meckel's Diverticula.—It is said that from a survey of the vast amount of literature and the enormous number of monographs upon the subject the unobliterated vitello-intestinal canal has been found in man in about twenty-four different forms and stages of obliteration. If one realizes that each form discovered can have some ten to twenty possible sequelae and complications, such as inflammation, gangrene, fistula, peritonitis, new growth formation, intestinal obstruction, tuberculous disease, etc., then one has only, by the simple mathematical procedure of permutation and combination, to see how many clinical forms Meckel's diverticula can assume to harass the surgeon. But as the disease has a constant origin irrespective of the sequela, it is put in a class by itself and will not be referred to further in this article.

Multiple False Diverticula.—The first group is the most interesting, as a common pathology exists for the disease in both small and large intestine. In the small gut the condition is rare; on going through 5000 case records in the Royal Victoria Hospital, Belfast, only one other case was discovered in addition to that operated upon by the writer, and the personal case is the

only one on record where not only the pre-operative but also the post-operative radiograms showed the condition. This was possible since all the diverticula had *not* been removed.

The case described is not an isolated example of a very rare disease, but rather a typical example of a disease well recognized but as yet receiving scant recognition in English text-books or by English writers in general. It is interesting to note that for diverticulosis of the large intestine the reverse holds good. It would appear to be a disease *par excellence* of the English-speaking people; certainly the best monographs on this subject are in that language.

Genuine cases of multiple diverticula of the small gut collected from the literature, when grouped together, form such a small collection that one is forced to realize that the condition is a rare one. Godard, who investigated the subject in France, believes that it is particularly rare in his country, and Moreau and Murdock writing upon the subject said, "Result of a post-mortem or surgical surprise—in all cases a disease deprived of all clinical interest—such for a long time was the total of our knowledge of small intestine diverticula."

Although the first case to be recorded in detail was in 1844 by an Englishman, Sir Astley Cooper, it is a noticeable fact that, excepting reports of individual cases such as those of Braithwaite and others, little work has been done upon the subject by Englishmen. In America J. T. Case has done some excellent work, especially from a radiographical standpoint, while in Germany Helvestine and in France Henri Godard have investigated the condition in considerable detail. It is an interesting fact that although Cooper described his case in 1844 in the post-mortem of a man who had died of cirrhosis of the liver, Chomel in 1710 had mentioned the first duodenal diverticulum, while diverticulitis of the colon, a much commoner disease, was not reported till 1849, and then by Cruveilhier.

The early cases were all found at post-mortem, but in 1914 Gordinier and Sampson discovered the first case in the living. The patient, a woman of 45, was suffering from intestinal obstruction due to kinking of the intestine at the site of a suppurating diverticulum. Until 1920 not a single case had been diagnosed by X rays, and in that year J. T. Case published two authentic examples. It is a noticeable fact that since then hardly six other cases have been so diagnosed notwithstanding the great improvement in X-ray technique.

Rarity.—The condition is a rare one. Until 1923 Sheppe had collected 30 cases from the literature of eighty years. Of the 26 collected by Watson, 18 were from post-mortems and 8 from operations. Godard collected 50 cases in all, and Case claims to have found 70, but some of these would be placed by other writers in one of the other subdivisions of jejuno-ileal diverticula.

Frequency.—The real frequency will never be exactly known, as so many cases exist unsuspected owing to an entire absence of clinical symptoms.

The small gut is often spoken of as the 'silent area of the intestinal canal', and when one sees upwards of five feet of jejunum surmounted on the mesenteric border by diverticula each as large as a hen's egg yet without symptoms, one realizes how true it really is.

Adams, writing in 1926 on the surgery of the small intestine, mentioned that he had never seen a single case.

Relative Frequency.—The relative frequency of diverticula of one part of the alimentary canal as compared with another is variously stated by different writers. The anatomist, pathologist, surgeon, and radiologist all give figures which differ considerably, as might readily be expected. Radiology is especially disappointing, as will be shown later.

Cases are on record, e.g., Stevenson's, where, although the gut was known to be covered with diverticula from the knowledge gained at a previous operation, yet repeated radiograms showed no diverticular shadows. Consequently it is easily understood that the radiologist's figures will underestimate the frequency of the condition. As regards the surgeon, his help is called in only in those cases diagnosed as such, or when the case, although unsuspected, is found during laparotomy. His figures will thus exclude all those—75 per cent of the total—who pass to their graves with the disease unsuspected. The anatomist's and pathologist's figures similarly will not give an accurate record.

From the above it is obvious how difficult it is to gather true statistics and how much one will vary from the other, although each in itself is a true and honest record. It will also help one to understand the variations in the figures which follow.

The frequency of diverticula of the alimentary tract is usually considered and is generally accepted as occurring in the following order of frequency: (1) Colon; (2) Meckel; (3) Duodenum; (4) Pharynx and œsophagus; (5) Stomach; (6) Jejunum. From this it will be seen that the disease, under survey is the rarest.

Larimore and Graham's figures show that in 3446 complete intestinal opaque meal examinations diverticula were found in 105 cases: 9 œsophagus, 3 stomach, 19 duodenum, 3 jejunum, 71 colon.

J. T. Case in 6874 complete examinations found: 85 duodenum, 4 jejunal, 1 jejuno-ileal, 138 colon. And later in 1920, after 10,000 examinations, he found 10 cases in all in the small intestine.

Reinhardt, in 5000 post-mortems, found 3 cases only, ages 66, 84, and 86. In all there had not been any digestive symptoms. Statistics from Johns Hopkins Hospital, Baltimore, show that in 2600 autopsies there were found 13 cases of diverticula of the small intestine; these presumably included duodenal, but excluded Meckel.

Henri Godard reports the result of 13,069 post-mortems collected from the Dresden City Hospital, Bender Hygienic Laboratory, and Boston City Hospital. There were, apart from 39 cases of Meckel, 16 diverticula of the small intestine. His figures are not far distant from the 1 per 1000 as quoted by others.

Marxer, in a personal series of 1000 X-rays, had 140 diverticula in all: 38 duodenal, 6 jejunal, 7 ileal, 6 appendicular, 83 colon.

The discovery of these cases can be divided into three main periods:—

1. Those discovered at post-mortem 1844–1914; this was prior to the period of safe abdominal exploration. The condition was thought to be a mere curiosity, and as presumably the patients had had few, if any, symptoms

it was looked upon as a matter of little importance. Reinhardt's 3 cases, aged 68, 84, and 86, had had no digestive symptoms.

2. The second period, starting in 1914, was when the disease was discovered in the course of laparotomy for some other condition, e.g., gall-stones, pyloric stenosis, obstruction, etc. (Gordinier and Sampson, the writer, et al.)

3. The third period, starting in 1920, is the present, when, with perfection of radiography and its technique, it is hoped to make a firm pre-operative diagnosis more often.

It will be thus seen that the disease should appeal to all branches of the profession, although the treatment of the acquired condition is pre-eminently surgical.

Moynihan, speaking of false diverticula, said, "the pouches may be numerous, rarely larger than a walnut, on the mesenteric side, and because of the large orifice do not as a rule retain food for long periods, and do not, therefore, often lead to serious complications."

Age Incidence.—As regards age, if one excludes one or two notable examples, the disease is one of old age, 70 and upwards. Case, in his series, found the average age for men was 66 and for women 60.

It is interesting to note that when complications arise they do so at an early age and are rarer in the older, so that whilst the uncomplicated cases were found chiefly post mortem and in the elderly, those cases with obstruction, inflammation, etc., appeared between the ages of 40 and 60.

The present case is exceptional in that the patient was 30 years of age.

Sex Incidence.—There is a preponderance of the male sex. In a series of 40 cases there were 29 males to 11 females. In fact, it is interesting to see in how many there was an enlargement of the prostate, leading in some to uræmia and death. The associated straining, with its resultant increase in abdominal pressure, was thought by many to be the *fons et origo mali*, but when one finds it in a female patient aged 30, as described here, one must look for other causes. In any disease found chiefly in men between the ages of 60 and 80 a co-existent hypertrophy of the prostate would exist, so that it is not safe to consider this necessarily a case of cause and effect.

Obesity was not mentioned in sufficient cases to allow of a definite statement. In the personal case recorded the patient outwardly was of average build, but her omental and mesenteric fat were very excessive. The leaves of the mesentery were separated by about $\frac{3}{4}$ in. of adipose tissue.

ANATOMY OF THE SMALL INTESTINE.

Prior to a study of the pathology of the condition, the anatomical features of the small intestine relevant to the present condition must be mentioned.

The small intestine is a long tortuous hollow muscular tube. Its average length is 22 ft. 6 in. in the male and in the female 6 in. longer. Extremes vary from a maximum of 31 ft. 10 in. to a minimum of 15 ft. 6 in. The outer wall is regular without sharp folds or sacculations. The diameter gradually decreases so that at the lower end it is only one-third of that at the beginning (Piersol). Four definite coats are easily distinguished—mucous (or lining), submucous, muscular, and serous (or covering).

The mucous coat contains glands, folds, and villi which aid its secreting and absorbing area. The epithelium covering the villi is a single layer of cylindrical cells which appear striated near the lumen. In many places over the villi mucus-producing goblet cells share the free surface. Lying between the ordinary epithelial elements migratory leucocytes can also be seen.

The stroma or tunica propria of the mucous coat resembles lymphoid tissue, being composed of a connective-tissue reticulum containing small round cells similar to lymphocytes. This stroma fills the space between the glands and forms the core of the villi over which the epithelium stretches. The deepest part of the mucous coat is occupied by a well-marked muscularis mucosæ in which an inner circular and outer longitudinal layer are distinguishable. Villi are minute projections of the mucous surface, barely visible to the naked eye. They impart a velvety appearance to the inner surface of the small intestine. They are less numerous in the ileum than the jejunum. They contain from one to three arteries, veins, and a lacteal.

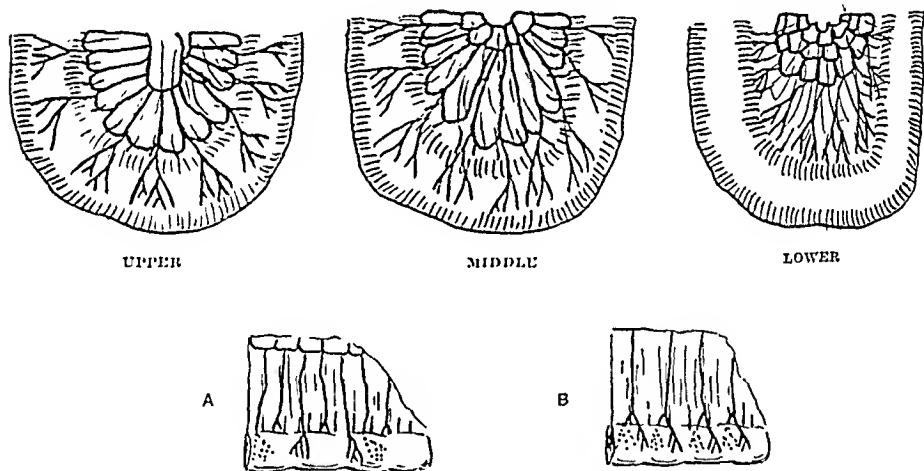


FIG. 118.—To illustrate distribution of blood-vessels to the small intestine, upper, middle, and lower portions. A, Peripheral terminations as actually found; B, As described in text-books.

The contents of the latter are propelled along by an involuntary muscle derived from the submucous coat.

The valvule conniventes are transverse folds extending partially around the gut. They are never obliterated even with distension. They are much larger on the attached side of the gut than on the free one. In the latter site they are often entirely absent, as also in the lower ileum.

The glands of Lieberkühn are simple tubular depressions found not only in the small but in the large intestine also. They are closely set, narrow, and extend through the thickness of the mucous coat as far as its muscular layer.

Lymphoid tissue is scattered in solitary nodules or in considerable masses as Peyer's patches, found especially in the lower ileum.

The submucous coat contains blood- and lymph-vessels and the nerve plexus of Meissner. It is lax, but not sufficiently so to allow displacement of the valvulae conniventes.

The muscular coat is composed of two layers. The inner circular is two to three times as thick as the outer longitudinal layer. The thin longitudinal layer, thickest at the free border, is often imperfect, especially at the attachment of the mesentery. The entire muscular coat diminishes in thickness from above downwards. The serous coat covers the intestine except at the point of attachment of the mesentery, where a bare area is left to allow blood-vessels to reach the gut. The bare area depends upon the amount of fat separating the leaves of the mesentery.



FIG. 119.—Arteries supplying portion of upper jejunum have been injected to show distribution.

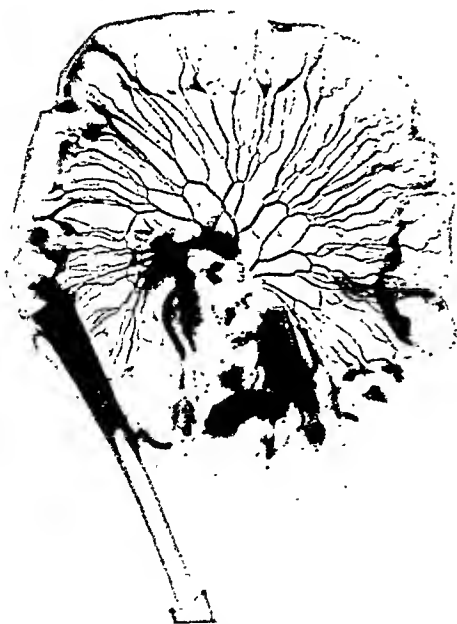


FIG. 120.—Similar to Fig. 119, but the intestine in this case is distended with oxygen.

The blood-vessels supplying the gut (Figs. 118-120) are always drawn in an artistic manner as forming a series of arcades (Fig. 118, B) from which terminal branches are given off, which would seem to bifurcate and to straddle the gut (as a rider would his horse), giving symmetrical branches to each side of the gut. Such in real life is not the case. The terminal artery leaves the arcade at a distance from the gut and passes to one side of it, and the next artery supplies the opposite side farther along, and so forth (Fig. 118, A). Frequently between them a small artery will be seen to supply the mesenteric edge.

Such an arrangement helps to explain the position

that diverticula take when once they are produced. They tend at first to bulge into the mesentery, but later fall over the intestine (*Fig. 121*) to one or other side, usually to the same side as the artery which causes their production.

The nerves supplying the small intestine are derived from the solar plexus, and consist of medullated and non-medullated fibres from the central nervous and sympathetic systems. After piercing the longitudinal muscle they help to form the intramuscular plexus of Auerbach consisting of both varieties of fibres and also microscopic sympathetic ganglia. The nerves continue obliquely through the circular muscle layer and form within the submucous coat the plexus of Meissner. From this latter non-medullated fibres enter the mucous membrane and form periglandular and subepithelial networks as well as supplying the muscular tissue.

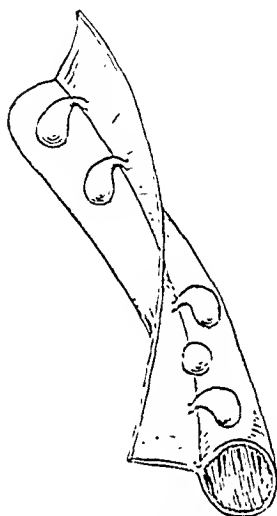


FIG. 121.—Diagram to illustrate diverticula on both sides of gut and into mesentery.

From a study of the anatomical features one is anxious to find what difference exists in jejunum and ileum, both as regards structure of wall and contents, to explain the frequency of these diverticula in the upper jejunum as compared with their paucity in the ileum.

Regarding contents there is little difference; in the jejunum the contents are somewhat more fluid, but in both places it is a fluid slimy stream which flows along. Regarding gas production, without obstruction and stasis this should not exist in the small intestine to any extent.

Comparing the lumina, one finds that the jejunum is three times larger than the ileum. Such decrease in diameter adds to the strength of the latter. It means that the same amount of longitudinal muscle fibres have to surround a smaller tube and therefore can give it a thicker coat.

In the jejunum in many cases the *longitudinal muscle is almost missing and certainly divaricated along the mesenteric border, and naturally in cases with much deposit of fat between the leaves of the mesentery this divarication will be greater still.*

Although the contents in the jejunum are greater in bulk and heavier in weight than in the ileum—two factors that would help to distend the gut and also to make it sag towards the pelvis, naturally exerting a great strain and pull upon the attachment of the arteries—the only anatomical factors worthy of consideration are the *great thinness of the longitudinal muscle* and the *greater size of the individual arteries supplying the gut*. This is constant and well marked.

The already weak portion is still further unsupported by peritoneum; thus, as a result, the circular muscle bears the whole stress of enclosing the bowel, and at the point where it is pierced by the artery with accompanying vein, fatty sheath, etc., a point of natural weakened resistance is produced.

PATHOLOGY OF DIVERTICULA.

The diverticula are thin-walled sacs. A section of the wall at the fundus of the sac consists of the mucous membrane of the intestine covered by the peritoneum forming one of the leaves of the mesentery. Between the two there may be some areolar tissue and fibrous tissue (*Fig. 122*). In most cases there is a marked deposit of fat encasing the sac—a similar finding to diverticula in the sigmoid. The presence of such fat is a difficult problem. Is it a primary condition and one of the factors concerned in the production of the disease? Has it, by increasing in amount, separated the mesentery, which separation in turn has caused a longitudinal divarication of the outer longitudinal muscle of the gut?

If fat is a primary condition, then why has the jejunum been affected where a deposit of fat is rare, while the ileum, where the mesentery is normally loaded with tabs of fat, is usually unaffected? Is the presence of this fat a precursor of hernia, just as it is in the inguinal and femoral regions, where, by dilating the canal and by subsequent traction, a track is formed for the future peritoneal protrusion? Again, in the heart, a diverticulum of the ventricle is subsequent to a heavy deposit in most cases, not only on the surface, but also leading between the fibres of the cardiac muscle. In diverticulum of the bladder and œsophagus the sac is usually encased with a thick deposit of adipose tissue. Or again, is the fat entirely secondary? Every surgeon has been impressed by the heavy deposit of granular fat around a pyonephrotic kidney. At the moment a gall-bladder is condemned because of opacity in its walls. This fat is said to represent past infection.

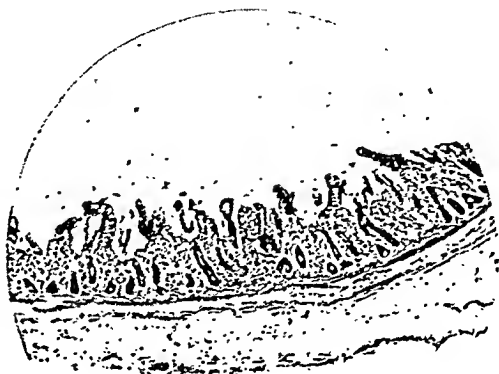


FIG. 122.—Low power. Section of fundus of diverticulum. Note absence of intestinal muscle, but muscularis mucosæ is still present.

Examples could be multiplied. No definite conclusion can be reached upon this point except that the character of the fat in those cases where it is primary is that of true adipose tissue—large clear cells with the nucleus in its typical circumferential position, compared with the granular fat found in the secondary cases, where a rich deposit of leucocytes and small round cells gives the fat a more elastic and resilient feel.

As has been pointed out, so many of the false diverticula have been without signs of inflammation that no cause has been given for the formation of a secondary deposit of fat.

Between the serosa and mucosa a variable amount of fibrous tissue may

be found. Its presence is a direct indication of inflammation in the sac, and as a result the serosa no longer moves freely upon the mucous membrane and a general hardness can be felt.

The origin of the sac is a projection, herniation, or protrusion of the mucous membrane at the mesenteric border (*Fig. 123*) of the gut through the substance of the muscle. As shown in the anatomy of the small intestine, the longitudinal muscle is strongest along the antimesenteric border, where peristalsis is greatest, and least along the mesenteric border. In addition, this area is unsupported by peritoneum, and if, by the deposition of fat, as is usual, the bare area is further increased and the muscle further weakened, either by pressure atrophy or by its fibres being divaricated through the fat passing inwards, additional cause will be found for herniation.

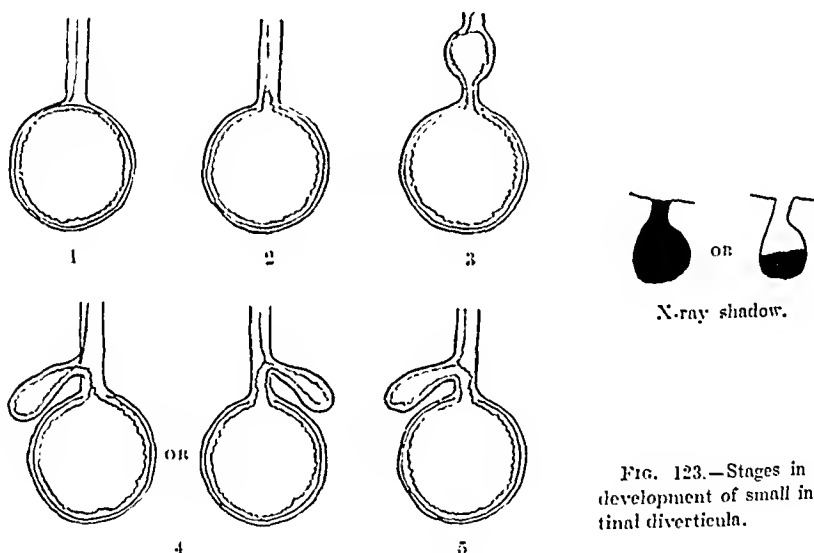


FIG. 123.—Stages in the development of small intestinal diverticula.

Such causes explain the weakness in the longitudinal muscle, which admittedly has weak points, but the circular muscle, which normally surrounds the bowel with an equal distribution of its fibres, must also be weakened. In this respect there is some definite relationship with the artery, because the sac occurs at the point of penetration of the artery, as can be seen in *Fig. 133*. Either a channel exists at the point of entry of artery and vein, and through this the mucous membrane bulges, in which case of course the circular coat would be pushed aside—in fact there would be a condensation of circular muscle on either side of the neck of the sac—or the pull of the artery on the wall or the drag of heavily laden ptosed bowel away from the fixed artery would pull out a conical tube of bowel, in which case it would contain all the layers.

In *Fig. 122* it will be seen that no muscle exists, showing that it is a genuine pressure rather than a traction diverticulum. Confusion should not arise over the circular and longitudinal muscle and the muscularis mucosæ. It is clear that even in false diverticula the muscularis mucosæ will be present.

In the larger sacs an occasional strand of muscle can be seen. This is only found in the cases where two separate diverticula have become fused, and the small band of circular muscle which previously lay between and separated them is carried over the top as a thin band, causing a dimple at the tip of the sac and making the fundus, instead of globular, somewhat heart-shaped (see *Fig. 125*).

That such fusion takes place is well recognized. In Boling's case, at the first operation the gut was seen to be studded with 250 to 300 diverticula, but at a second operation, owing to fusion only 53 were present although individually the average size was greater. Naturally the strap of muscle between would now be represented upon the sac as an atrophied band. At first appearance the specimen might be mistaken for a case of pneumatosis of the small intestine. This rare condition, described by Nitch and later by Pugh, includes the presence of air sacs—large and small—in the mesentery, either close to the wall of the gut or passing towards the root of the mesentery, and gives the feel of surgical emphysema. The causative force is not known; possibly it is a sequela of aerophagy. There is no connection, however, between pneumatosis of the mesentery and false diverticulosis.

In size the sacs vary—anything from a pea to a small orange or apple. In the writer's case the average size was that of a golf ball. The projection, although always on the mesenteric side, is not absolutely mid-line. As the sac enlarges it grows between the layers of the mesentery, but sooner or later tends to bulge to one or other side (see *Fig. 121*). This is either: (1) Because the sac is following the particular artery which has been the determining factor in its production, and this may be on one or other leaf of the mesentery; or (2) For purely mechanical reasons, as it is easier to accommodate the growing sacs in a given area, if they take up a position alternately on either side of the bowel instead of being side by side. In Berry's case the majority fell to the right side of the gut. He considered that owing to the obliquity of the mesentery there was greater room on the right side than on the left to accommodate them.

Mention has been made of the relationship of the sacs to the straight terminal arteries which supply the bowel arising from the terminal arcade. The relationship of these vessels is definite and constant and is the only incontestible factor in the etiology of the condition. Hensemann was the first to point this out, and proved his point by serial sections.

The sacs in the uncomplicated cases are empty or contain some fluid chyme intermixed with air. Inspissation of the contents is rare for many reasons—the contents are very fluid, stasis is rare, the stoma is large so that ingress and egress to contents is easy, peristalsis in the small intestine is rapid, etc. In the literature the presence of inspissated 'faeces', gall-stones, and malignant growths is mentioned in individual and isolated cases, but judging by the large proportion of sacs in individual instances—up to 500 in some—and the number of cases without symptoms, it can be readily realized that stasis and other complications in these sacs are relatively rare.

Naturally complications do arise from time to time, probably owing to the enlarging of the sac and to the narrowing of the neck. In the early stage the sac is small and the stoma is large in proportion. As long as the sac

lies in the mesentery, naturally the opening into the bowel will be at its lowest point, and with the patient upright there will be good gravity or dependent drainage. Later, when the sac hangs over the edge of the gut, the neck gets lengthened out and narrowed, drainage is uphill, and stasis will naturally ensue. A typical example of fecal stasis is shown in Watson's case. This complication is the exception in small-intestine diverticula but almost the rule in the colon.

Hirschmann reports a case of cancer starting in the diverticulum, and Holt a case where a sarcoma and diverticulum are associated. There is no definite evidence to show which is the primary factor. Diverticulitis and peridiverticulitis are the usual complications leading to localized or generalized infection. Kinking and obstruction are very apt to ensue in such cases, e.g., those of Gordinier and Sampson, Braithwaite, Kaspar, the writer, and others. Infection around the diverticulum is more frequent in the colon or duodenum. Molesworth reports in this connection an interesting case where there was associated with the duodenal diverticula considerable diverticulosis of the sigmoid. The patient died of pancreatic necrosis secondary to inflammation of the duodenal diverticulum.

The emptying of the sacs is dependent upon two factors: (1) gravity, and (2) the contraction and approximation of the leaves of the mesentery. This last factor is only free to act as long as the sac is in the mesentery, as in the early stages. But later when it protrudes through the anterior or posterior layer (see Fig. 123) it will no longer be so compressed. The question of fat again arises. The sac can only be compressed if the two leaves of the mesentery can be approximated, and this naturally is impossible if a thick layer of fat is sandwiched between.

The absence of muscle in the wall of these sacs means that they are unaffected by the general peristalsis of the gut. The suction or *vis a fronte* of the stream as it flows past may empty them, but they have no driving force or *vis a tergo* of their own. The absence of muscle in the wall also results in the absence of colic, which is a marked feature of all 'true' diverticula.

In the early stages gravity is sufficient, but later, with kinking of the neck, drainage is rendered more difficult and a feeling of fullness and distension exists, and if no adhesions are present the sac can only empty by the patient's changing from the standing to the lying position. This explains the comfort experienced in many cases—the writer's patient invariably got relieved of this distension by lying down. With occlusion of the neck, stasis, putrefaction, and gas production are natural sequelae, clinically manifested by borborygmi, distension, and flatulence.

Regarding the situation of the diverticula, they may arise at any point from the duodenojejunal angle to the ileum. When arising at the flexure they cause trouble both as regards diagnosis, in that they often simulate a Haudeck's niche (Hurst, *Guy's Hospital Reports*, 1932, March—see Fig. 130), and also in their surgical treatment. The largest of the sacs are usually at the proximal portion, but this rule is not absolute, as often one finds at the beginning a series of large sacs followed by smaller ones, but scattered through the latter at irregular intervals many large ones.

The number is never constant. It may vary from quite a few to perhaps 500 to 600. There is nothing constant about the cases which give rise to symptoms. They may arise in diverticula near the duodenojejunal flexure or in ones farther down, and may occur in patients with few diverticula as readily as those in whom the small intestine is just studded. They do seem to occur more readily in the giant diverticula than in the small ones. The sac wall is generally very thin. At operation the fundus can be invaginated with the finger right into the lumen of the intestine with great ease.

THEORIES OF CAUSATION.

For many years it was thought sufficient to classify diverticula into congenital and acquired, or true and false, the latter terms depending upon the presence or absence of muscle in the wall of the sac.

Kausch was the first to point out that 'congenital' and 'true' and 'acquired' and 'false' were not synonymous terms. Brandes reported a case of acquired diverticula which were 'true' in that all the wall was present. At an early stage it was realized that these diverticula were an acquired condition for many reasons: (1) never found in the fœtus, (2) never found in the young, (3) a disease of the retrogressive period of life, (4) incompatible with embryological growth, (5) study of the progress of the disease within the same patient, e.g., Boling, Stevenson, et al., (6) their structure, (7) their inconstancy in size, shape, appearance, situation, (8) absence of other congenital defects, etc.—in fact, no brief could be held in favour of the congenital theory. On the acceptance of the disease as an acquired condition many theories have been produced to explain its occurrence.

Roth in 1872 formulated the idea that it was a fatty degeneration of the tunica muscularis. But why should it attack jejunum rather than ileum? The patients are not necessarily obese, some are young and healthy (the writer's case, age 30). Histologically there is no proof that the longitudinal muscle in the upper jejunum has alone been involved in a fatty degeneration.

Klebs in 1869 pointed out that there was an artery in relation to the sac. He was of the opinion that the cause was the drag of a heavy gut away from the fixed vessels, with the formation of traction diverticula. Such diverticula are all true and contain all the layers of the gut. It is obvious that a sac that falls over the edge of the gut cannot have an artery exerting tension upon its fundus.

Hansemann in 1899 examined carefully the relationship of the artery to the sac, and by serial sections was able to show that the pouch appeared *beside* the artery, and that, although the artery did not draw out the sac, the sac made use of the channel so formed to hudge through the muscle. He considered the causative force was increased intestinal pressure from gas or faeces. In support of his thesis he produced diverticula by inflation.

Chlumsky in 1899 repeated Hansemann's experiments, and dilating the gut found it ruptured at the antimesenteric border, and concluded that the condition was not due to increased intestinal tension.

Della Palma Modesto blew air into gut deprived of its peritoneum. In one case only did a diverticulum appear, and in this the patient had died of

tuberculosis. He concluded, therefore, that this disease is only produced in the intestine of people suffering from some degenerative disease, e.g., tuberculosis or old age, or else that there is a congenital predisposition.

Gisbertz in 1929 mentions this fact when he reports the case upon which he operated under local anaesthesia. The patient was suffering from tuberculosis of the lungs and glands of the abdomen. He holds that each gut has a tendency to form diverticula and each intestine is a law unto itself. The appearance of a sac depends upon the hole in the wall through which the vessels pass. He believes that external circumstances are the exciting cause; occupation, mode of life—in fact anything which modifies the peristalsis of the gut.

Graser (1899) thinks that venous congestion, e.g., portal obstruction, is the deciding factor. The distended veins, by enlarging the opening in the muscle, produce a *locus minoris resistentiae* and, as a result, hernia formation. He found supporters in Akerlund, Oelmeel, Moreau and Mardock, Della Palma Modesto, Wakanaba, Buchni, et al. They held that the disease occurred almost exclusively after the age of 40; it was the period of circulatory embarrassment and failure.

Sudsiki in 1900 produced the theory that, rather than the vein actively distending, it was allowed to do so passively by the diminished resistance of the connective tissue.

Davies-Colley considered that the fat around the vessels and between the muscle fibres was the cause. It acted by causing a weak spot in the muscular tunic, or by its pressure causing the muscle to atrophy, or by infiltrating the muscle reducing its resisting power. This helps to explain its position in the mesenteric border in the small intestine and in the region of the epiploic bodies in the large. Such a theory is not tenable. Fat is much more heavily present in the ileum. It is well recognized that the epiploic bodies have no part in the causation of sigmoid diverticula. They contain the diverticula because they contain the artery, and the artery is the conductive force determining the direction that the diverticula will take.

That 'atrophy of muscle' must play an important part is stressed by many, and Neupert reporting upon a case considered 'muscle weakness' to be the primary cause. Boling in his article considers lack of 'muscle tone' the most vital factor.

Traction diverticula arising from tumours, myomata, hæmangiomas, lipomata, tuberculous glands, etc., are frequently quoted, but no serious attempt can be made to correlate such cases with the condition under investigation.

General cachectic states resultant upon tuberculosis, typhus, chronic osteo-arthritis of the spine, carious teeth, etc., are said to be more prevalent in those with diverticulitis than in an equal number of individuals of the same age used as controls.

Although Keibel and Mall and Lewis and Thyng have shown the presence in the very early fœtus of islands of isolated cells which normally atrophy, but which by persisting have produced cysts and diverticula, no connection can be claimed between them and the diverticula under examination at the moment. Such diverticula or cysts are included under congenital diverticula of enterogenous origin.

Nitch described in detail the condition of cystic pneumatosis of the mesentery in which there are localized 'gas bags' in the mesentery. This, although interesting and clinically somewhat similar in appearance to multiple diverticula, cannot be considered as an etiological factor.

In 1923 Helvestine, discussing the condition, concluded that no single factor could explain the disease, and decided that the three following factors working together were necessary: (1) Traction by mesenteric vessels or following adhesions; (2) Degeneration of intestinal musculature; (3) Increased intra-abdominal pressure. One cannot agree with this:—

1. Firstly, adhesions can be shown to be absent in all sacs unless inflammation has taken place, and in such cases the adhesions have an inhibiting influence rather than otherwise upon the growth of the sac. Traction by mesenteric vessels does not occur. If so, one would expect to find a vessel attached to the fundus of the sac and the artery taut; also the fundus of the sac would consist of all the coats and not mucosa only.

2. Degeneration of intestinal musculature: Why should this involve the longitudinal muscle only? Why only that portion in the mesentery? Why only in the jejunum and not in the ileum?

3. Intra-abdominal pressure: Such probably was considered as a possible cause from the fact that many of the patients had died of enlarged prostate with probably vesical tenesmus, many had inguinal herniæ, and many were constipated. It has, however, been pointed out on the other hand that many had lax patulous abdomens, and, as in the writer's case—a woman of 30—little straining of any sort could be proved.

Sheppe modified Helvestine's views slightly and gave as the three factors: (1) Rectal or vesical tenesmus; (2) Atrophy of inner circular layer of intestinal musculature; (3) Traction of mesenteric vessels due to sclerosis and adhesions. Of these, the second is especially worthy of note. Degeneration of the circular muscle would surely produce a circular bulge involving the circumference of the gut over the area involved, and could in no way explain a series of bulges involving the gut in its longitudinal direction.

Any theory propounded to explain the origin of these diverticula must, if correct, embrace the following facts: The disease occurs in both sexes, usually after 40, but it may be found in patients as young as 30; the upper jejunum is usually affected and the larger diverticula are seen nearer the duodenum; there is a definite relationship with the artery, and in several cases a stricture further down.

A fact overlooked by all writers, and one which seems of paramount importance, is the *anatomical disposition of the longitudinal muscle in the upper jejunum*. It is thinned out, wasted, or almost missing owing to the fact that the jejunum has a three times greater circumference to be covered than the ileum. In fact, one might almost say that just as the colon has three longitudinal tæniæ, so the jejunum has longitudinal tæniæ on three sides only. This congenital divarication can be still further accentuated by separation of the mesenterial leaves by fat. The circular tunic is the only one left to protect the gut, and it in turn is pierced by the artery and veins to the gut, giving a potential seat of lowered resistance. In addition the individual arteries to the jejunum are larger than those to the ileum. The further

factor needed is increased pressure such as is found in stricture of the gut (Godard, Jefferson, Cooper), constipation, vesical straining, coughing, etc.

Experimentally the production of diverticula occurs at these situations by increased intra-intestinal pressure, and the hernia so produced is a false one analogous to the sac found clinically.

One must conclude that: (1) Increased intestinal tension, (2) acting along the channel for the artery, (3) at the point where the longitudinal muscle is divaricated, is the causative agent.

CASE REPORT.

The following personal case helps to exemplify the condition.

Mary B., a spinster of 30 years of age and a shop assistant until three years previously, was sent from the country to hospital with a diagnosis of perforated peptic ulcer. Seen soon after arrival her abdomen was tender in the epigastrium and she had vomited; but beyond slight elevation in pulse and temperature nothing definite could be discovered. Her abdomen was not rigid. Liver dullness was present. It was fairly definite that she had not had a perforated ulcer, and as she had a history of three previous attacks it was decided to wait.

The following day she had improved greatly and had an opaque meal examination which showed "multiple obstructions in the small intestine, ? Adhesions, ? T. B.

peritonitis". Two days later she developed acute complete small-intestine obstruction, and she was operated upon at once. The upper jejunum laden with fat was studded on the mesenteric border with large diverticula; some lay between the layers of the mesentery, but others fell over the edge of the jejunum, some on one side and some on the other.

At one point a hard mass was found in the mesentery from which pus was escaping, and at that point the gut was angulated and obstructed. Rapidly and carefully to prevent peritoneal soiling the writer excised 4 ft. of the small intestine about 6 in. from the duodenojejunal flexure, and including, it was thought, all the affected gut. At the time further examination of the abdomen was impossible as the intestine had to be packed away with meticulous care. Although at the time it was thought that all the diverticula had been removed, subsequent radiograms showed that this was not the case. The patient made an uneventful recovery and since has remained perfectly well.

The most noticeable features were the enormous deposit of fat, the thinness of the

walls of the sacs—with the finger the fundus of each could be invaginated with ease into the lumen of the gut—their enormous number, their individually large size (average was that of a golf-ball), and the relative absence of grave symptoms previously. There were signs of old scars, and from her symptoms it would appear that she had had either three previous attacks of peridiverticulitis without perforation which had settled down, or perforation with localized abscess, which had drained into the gut and had not given rise to adhesions and obstruction as on the present occasion.

Fig. 124 gives an idea of the macroscopic appearance of the bowel removed at operation. The excised intestine was filled with bismuth solution, and the



Fig. 124.—Sketch of bowel resected.
Seat of perforation is easily seen.

radiogram taken (*Fig. 125*) gives an exact picture of the relationship of diverticula to jejunum. It will be noticed that the small diverticula have a round fundus, the large ones are depressed and dimpled owing to the strap of circular muscle mentioned previously. The resected specimen measured 4 ft. in length, and the upper end started 6 in. from the duodenal flexure. There was sufficient tissue to allow a satisfactory side-to-side anastomosis to be performed.

Fig. 125, A shows the point of perforation of the diverticulum. Since several of the diverticula lay behind the intestine, the outline is masked by the bismuth in the latter. During the convalescent period further X-rays were taken and it was rather a surprise to find further diverticula still present. With the co-operation of a willing patient a series of these was taken hourly for ten hours, and, as a result the appearance of diverticula at different times and in different stages of emptying has been obtained (*Figs. 126-128*). Some of these show the typical pyriform or globular mass hanging from the intestine. In others, where the sac contains the bismuth below with gas above, there is the saucer-shaped shadow with a more or less horizontal upper surface.

The patient's previous history showed her to be a strong healthy girl up to three years ago—fond of tennis, rowing, walking, etc.



FIG. 125.—Radiogram of resected specimen filled with barium. Note diverticula present along the concavity—mesenteric border. A, Perforation.



FIG. 126. Five-and-a-half hour picture. Globular shadows distinctly seen.

Since then she had had intermittent ill health; this included a thyroidectomy two years ago.

She complained of upper abdominal pain, pain after food, and even before food, though pain was occasionally eased by light food. The pain did not radiate and it had recently become almost constant. Its usual situation was slightly above and to the left of the umbilicus. On a few occasions she had had night pain, and had given up meat, potatoes, and heavy food. Her pain suggested in an indefinite way a peptic ulcer. There was no vomiting as a rule, but flatulence and borborygmi were a marked feature. From being a strong healthy girl she had been forced to give up work altogether.

During this period of three years she had had three very acute attacks. In the first, which was diagnosed 'acute gastritis', the vomiting was so excessive that stomach lavage was resorted to upon several occasions. The second attack was diagnosed 'peritonitis'



FIG. 127.—Note three diverticula hanging from intestine (not filled). This was taken four and a half hours after ingestion of the barium meal.



FIG. 128.—Ten-and-a-half-hour picture. A diverticulum can be seen in right-hand upper quadrant of radiogram opaque in lower half, gas in upper portion.

—sudden severe abdominal pain, with abdominal rigidity, etc. She was carried home and kept in bed for two months. The third attack was like the second; she was admitted to hospital and prepared for operation, but as the attack subsided, operation was postponed and she was eventually discharged.

On physical examination there was little of interest. The abdomen moved freely; there was general tenderness and muscle guarding, but no tumour and no free fluid. Liver, kidney, and spleen were not obviously enlarged. There was no rigidity. Rectal and vaginal examination were negative. In fact, from her symptoms and signs no definite conclusion could be reached.

SYMPTOMS.

It has been found impossible to draw up an exact clinical picture of these cases with exact text-book signs and symptoms. Such naturally is the case in a disease which at the one extreme may be symptomless, and at the other may be producing symptoms of the severity of acute intestinal obstruction. In the following description of the symptoms an attempt has been made to collect and correlate any feature that seems common to all the cases so far recorded in the literature.

Duodenal diverticula are not so serious as colonic, and jejuno-ileal diverticula are much less serious than either. Just as these three areas vary in the gravity of their prognosis, so one finds the vagueness of their symptoms bearing a direct relationship. Fully three-quarters of all the recorded cases were without symptoms, and in the remaining quarter the symptoms varied greatly, depending upon whether the patient had a long period of chronic abdominal ill health or was first seen as a surgical emergency.

Symptoms depend upon closure of the neck with resulting inflammation and the production of a closed cyst. In the chronic case the symptoms resemble a peptic ulcer in a general way, but, as Frank Smithies pointed out in 1920, there is no clear-cut dyspepsia and certainly no periods of remission.

Pain is a marked feature, but never very severe, never colicky in character like Struther's case, where the diverticulum had a muscular wall, and, as a result, was capable of producing a pain of intense severity. The pain is chiefly epigastric, and passes to the left of the umbilicus and even down the left side of the abdomen. It varies with the situation, and is due to distension of the sacs. Case, operating upon a patient under local anæsthesia, was able by compressing the distended sacs to produce a pain which the patient agreed was similar to the original one. Pain in the early stage comes on half an hour to three hours after food, but in the advanced stages it is present almost all the time. Food, fluids, or alkalis have little or no effect upon the pain. Greatest comfort is obtained by lying down.

Pain is greatly aggravated by coarse food, potatoes, and acid meals. In this respect these cases greatly resemble the series of 34 duodenal diverticula described by Oehnel and Akerlund. Such also is found in duodenal ulcer, and several patients have had a history of fifteen to sixteen years with the pain worse after meals, and with a radiation to the shoulder. Shoulder-pain is not a feature, but many of the cases are complicated by an additional disease, e.g., gall-bladder trouble, etc., the symptoms from which may tend to mask the original condition.

Probably the most constant and most interesting feature of the disease is the flatulence, borborygmi, gurgling, and rumbling that all suffer from. In Usland's case the patient described his abdominal sensation as being like 'clothes hailing in a pot', and in Braithwaite's case the patient had to retire to prevent his confusion. All complain of distension, and this was much greater with standing than lying, and was eased usually by eructation of foul gas or sour fluid. The meteorism and weird noises, being such a feature, are one of the helpful points in differentiating the disease from a peptic ulcer. The age factor must also be considered. The ulcer history has usually started before 40, whilst diverticula are rare till considerably after that. Effect of alkalis and periods of remission also help, but in most cases diagnosis is difficult, and when the two diseases are coexistent, as is not infrequent, a definite diagnosis clinically is impossible.

Vomiting is not constant—it was a marked feature in some cases, e.g., those of Rothschild and Case, but variable with others. Nausea is more frequently found. Constipation is the rule rather than diarrhoea, as compared again with duodenal ulcer, where the hypermotility in the colon usually ensures a regular movement of the bowels. Two cases vomited blood, but this is not mentioned in the others. In one diarrhoea and constipation alternated. 'Mild prolonged indigestion', 'bilious attacks', 'attacks of auto-intoxication', loss of appetite, coated tongue, muddy complexion, and general debility, are mentioned in many of the cases.

Loss of weight is quoted by some as an important feature, e.g., Hunt and Cook's patient had lost 3 stone. MacKeechie's patient was very emaciated, but others, e.g., Rothschild, point out specifically no loss of weight, and the writer's case had actually gained. One feels that any change in weight in these cases is not the result of the disease, but depends upon the liberality or otherwise of the diet permitted.

That melæna is a marked feature is stressed greatly by Continental writers, especially the Scandinavians. The patients of Hunt and Cook, Rothschild, and MacKeechie all showed this condition. Usland in his case was able to show a venous plexus in one of the sacs actually bleeding at the time of operation. The bleeding was very severe in Hartung's patient.

Great care is required to separate the symptoms of the diverticula proper from those of some other coexisting disease. In 1921 Terry and Mugler collected 19 cases of jejunal diverticula. Of these one occurred at the duodenojejunal flexure, but in only 3 were the symptoms attributable to the diverticula.

The fractional test-meal is reported by Oehnel to have failed as a clinical aid to a diagnosis, but as any one writer gets so few examples of such a rare disease, no sufficient series can be obtained on which to form a definite estimate of its value.

There is no constancy in the coexisting diseases. Gisbertz' case had tuberculosis of the lungs and abdominal glands; many of the early cases had uræmia, others duodenal ulcer, gall-stones, etc., so that no definite causal relationship could be established.

The above series of symptoms merely accentuate the fact that no clinical syndrome is known to include all the cases. The very vagueness of the condition is one reason for adding to its interest.

RADIOLOGY.

In 1914 Forssell (radiologist) discovered by barium meal, and Key (surgeon) confirmed by operation, the presence of a duodenal diverticulum. This was the first recorded case of a definite pre-operative diagnosis in the history of the disease. It was not till 1920 that Case (Battle Creek) was able to find the first jejunal diverticulum and to confirm it by operation. In an excellent article he described 5 such cases. The radiological evidence cannot be doubted, but only 2 had subsequently laparotomy to verify their presence.

In the somewhat similar cases of Boling and Stevenson, the abdomen was opened and intestines with 150 to 250 diverticula were seen; no resection was performed, and yet in both cases no sign of the diverticula appeared radiographically although repeated X-rays were taken with the knowledge that the diverticula were present. Braithwaite and Berry had positive results in their cases only after difficulty. Heidecker in 1926, and Tengwall in 1930, also claim positive X-ray results, the latter only after repeated pictures had given negative results.

In 1924 Baastrup reported a case where the diagnosis had been made after X-ray examination. His case, although interesting, was a single traction diverticulum due to the presence of softening tuberculous glands, and as such should not be included under multiple false diverticula.

Gisbertz in 1929 reported an interesting case. The operation, although performed with great care under local anaesthesia, was unsuccessful. The chief interest lay in the pre-operative X-ray diagnosis. C. Orbaan also records in a Dutch paper a case diagnosed radiologically. Such a small list makes one realize either how difficult the cases are to radiograph or how necessary is some further improvement in technique.

In the writer's case the shadows seen in the pre-operative picture were recognized only *after* operation as diverticula, as the eye then knew what was to be expected. Incidentally the condition was unsuspected by the writer. In the series of photographs taken after the operation the shadows assume the typical appearance. This series is unique in that it shows diverticula at hourly intervals and in different degrees of fullness. Case, describing his radiograms says, "the findings present an interesting relief from the monotony of everyday life".

Infinite patience on the part of the radiographer is required in taking the series of photographs, in timing the picture correctly, and in placing the patient at the correct angle. In reading the radiogram considerable skill and knowledge of the normal and an appreciation of super-position are required. A large meal by its bulk will obscure the picture and by its weight will drag the stomach down into the pelvis if the patient is standing, or into one or other of the paravertebral grooves if the patient is supine. Hence a small meal.

A series of pictures at hourly intervals will show the constancy of a shadow, and if two films are superimposed, the fixity of the part can be seen. A twenty-four hour picture often will show the shadow still present; the lack of muscle in the wall of the diverticulum naturally means very sluggish emptying.

A preliminary X-ray without bismuth is recommended in all cases in the hope that the small sacs will be seen filled with air or gas, and by the shape of the rounded margin a diagnosis be made. Such has never been successful. With a barium or bismuth meal the typical shadow of a diverticulum is either a round or pear-shaped shadow communicating with the gut. As the latter is usually empty, a series of such shadows side by side is typical (see *Fig. 127*).

The other form that the diverticulum may take is that of an opaque semicircle or D (with the straight line horizontal) and above this a similar shadow filled with air. The two portions are separated by a horizontal line, similar to what one would expect to find from a balloon partially filled with a heavy fluid below and gas above. *Fig. 129* shows the condition diagrammatically. When such a picture is positive a definite diagnosis is simple, but it is disheartening to see intestine studded with diverticula and yet with radiography to obtain a negative result each time.

There are two or three possible explanations of this:—

1. The diverticula are still in the mesentery, i.e., attached to the *upper* margin of the gut, and hence do not fill; filling in such cases could only be obtained by placing the patient in the Trendelenburg position.

2. The diverticula, either large or small, are *already* filled with some other medium which must first be displaced; abdominal massage might help to fill the sacs. Such a possibility is rare, as at operation the sacs are usually entirely or partially empty.

3. The diverticula have become so large that, hanging down on either side of the gut, the neck is kinked and entrance is cut off. This also is unlikely, as the stoma will usually admit one finger and even two with ease.

The writer's case is an example of the disappointing results of X rays. *Fig. 125* shows the 4 ft. of intestine removed and filled with bismuth, and yet *Fig. 127*, taken before operation, gives no indication of the presence of diverticula to such an extensive degree.

With a diverticulum at the duodenojejunal flexure, difficulties arise. First, on an antero-posterior view the stomach, if filled with an opaque meal, may entirely mask the condition by covering up the smaller shadow. Secondly, a perforating gastric ulcer may produce a Haudek's niche almost identical in appearance to the duodenojejunal flexure shadow. This has been pointed out by De Quervain, by Akerlund at considerable length, and recently (1932) by Hurst in *Guy's Hospital Gazette* (*Fig. 130*). The first difficulty can be overcome by a true lateral or partially

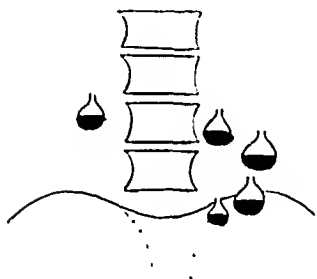


Fig. 129.—To explain the saucer-shaped shadows. (After Godard.)

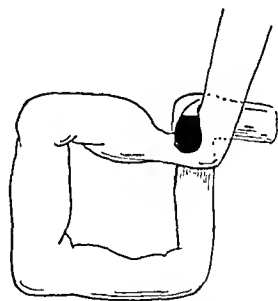


Fig. 130.—To illustrate the difficulty in differentiating the shadow of a Haudek's niche from that of a duodenojejunal flexure diverticulum. (After Hurst.)

lateral picture; but the second difficulty can only be overcome by screening the patient. A Haudek's niche fills at once from the stomach meal, a duodenal or duodenojejunal flexure diverticulum is only seen *when* the meal has passed through the pylorus. A further picture taken after emptying and even up to twenty-four hours may still show a shadow. This may happen in any of the conditions and is of no value in making a differential diagnosis.

It is interesting to note that just as jejunal diverticula is a rare condition, its X-ray diagnosis is very much rarer. In duodenal diverticula the reverse holds good. It is diagnosed much more frequently in the X-ray room than in the operating theatre.

The most frequent errors in reading the radiogram of jejunal diverticula consist in considering the condition one of: (1) Gastric ulcer with Haudek's niche: (2) Generalized adhesions—tuberculous or otherwise; (3) Ptosis.

TREATMENT.

It is generally agreed that in 75 per cent of all cases the disease is symptomless. It thus passes undiagnosed or even unsuspected and is only found fortuitously at a post-mortem examination. In such cases the question of treatment naturally does not arise. In the majority of the early recorded cases the patients had died in old age and had spent a life free from major abdominal discomfort. The ages in Reinhardt's three cases were 64, 84, and 86. In all three there never had been any digestive symptoms at all.

A more difficult problem is the case where the condition is discovered during laparotomy for some other disease, e.g., gall-stones (Rothschild) or duodenal ulcer (Usland), or where a diagnosis has been made during a routine intestinal opaque meal examination.

Take the latter case first. Is it fair to subject the patient, usually elderly, to an operation which is not easy to perform, which is attended with considerable risk, and which, when the abdomen is opened, it may be impossible to complete satisfactorily? When one realizes that the majority pass through life without complications and that complications when they do occur have rarely been fatal, one cannot hesitate to postpone operation, with the understanding from the patient that he will keep under regular supervision every six months and report progress if any.

The former case is different. The abdomen is open, and it is now a personal matter for the surgeon. With a relatively young patient, with a competent surgeon, with diverticula localized to a limited area of intestine, resection of the affected portion probably is the correct procedure. If this is successful a perfect result may be expected, but in all cases a careful examination of the intestine below must be made to exclude a stricture. Such is found to exist in many cases, e.g., Sir Astley Cooper's original case; Henri Godard discovered a contracted scar in the ileum, where the patient had had the intestine caught in a strangulated femoral hernia. Geoffrey Jefferson was forced to do a double enterectomy not only of the area affected with diverticula but also the constricted portion below.

In all cases where symptoms, even if mild, have definitely originated from the diverticula, operation is advisable. In at least two cases the

abdomen was opened when the symptoms were mild, and when the condition was discovered it was thought to be inoperable and nothing was done. In both of these the patient returned later with aggravated symptoms. Enterectomy was performed with complete success.

In Stevenson's case the patient returned after two years, and several feet of the intestine with 154 diverticula were removed, with complete cure. Boling's case was interesting in that at the first operation there appeared some 250 diverticula, but at the second operation when a successful enterectomy was performed there were found to be only 53. Obviously fusion and coalescence had taken place to reduce their number.

In all cases where the diverticula are causing symptoms, even mild, operation is advisable, as it is noticed that practically all the patients in whom a major calamity, such as perforation or intestinal obstruction, supervened later had had a definite previous history.

Regarding the operative measures available in the uncomplicated case, the technique of choice is complete removal of the affected area with restoration of the continuity of the gut. In the writer's case the junction was of the side-to-side variety, as it was considered that the stoma in an end-to-end union might, on healing, contract and produce a slight contraction ring or stricture. This probably would not be sufficient in the normal individual to cause symptoms, but in a patient with a tendency already to form diverticula it might be the starting-point for further trouble. This is probably a small point, as most of the cases where technique was stated had an end-to-end anastomosis and the result seems to have been excellent.

At the upper end difficulty often arises, as the jejunal diverticula may be represented at the duodenojejunal flexure by other diverticula and even the duodenum itself may be involved. As a result healthy intestine cannot be obtained. This difficulty has been overcome by Rothschild and others by performing a gastrojejunostomy and using the opening into the diverticulum to form the new stoma between stomach and jejunum.

In other cases, e.g., Braithwaite's, where resection was at first considered impossible, a short circuit was performed. This, like all operations devised for two purposes, was a failure. If the short circuit works well, the loop does not drain, and if the loop drains well, the short circuit does not function. In Braithwaite's case ultimately a resection was performed with good results. Minor operations, such as removal of individual sacs, plecting, infolding, strengthening of the wall, etc., are recommended; but when perhaps up to 500 sacs may be present, and where the appearance of the sac is really a result or symptom of the disease, such operations naturally will be left for the theoretical surgeon and for the text-books. Such treatment is indicated in the single diverticulum of the congenital variety, such as Meckel's, where amputation with infolding gives a permanent result.

The extent of small intestine that can be resected without affecting the health of the patient has not definitely been decided upon. It must be remembered that jejunum is more valuable than ileum on account of its absorbing surface. Adams estimated that 7 ft. of jejunum or 12 ft. of ileum could be removed without disturbing the patient materially. In 1910 Bremen in a man of 61 removed 17 ft. 9 in. of small intestine. The patient lived two and

a half years and died subsequently of marasmus due to failure of fat absorption. The normal average length of small intestine is 22 ft., but the extremes vary from 15 to 30 ft., so that the important factor is not how much is removed, but how much remains. In animal experiments it is found that in dogs 50 per cent can be removed with safety. In such cases it is found that villi and mucosa hypertrophy considerably. As a general rule in man, if more than two-fifths of the entire small gut is involved, resection may be considered impossible.

It is readily realizable that spontaneous cure or recovery is impossible in these cases which are due to the absence of muscle in the wall of the sac. With all mucous herniæ, where the distension of the gut and sac acts as a *vis a fronte* distending the sac, and the adhesions on the exterior act as a *vis a tergo* preventing recoil, the disease is progressive. Thus in the case of those patients in whom operation is refused or contra-indicated, some regular mode of life must be advocated. In many cases the symptoms closely resembled a duodenal ulcer, and the restricted, alkaline, carbohydrate diet with which they were treated seemed to suit. All complained that rough food, potatoes, and meals with a residue did not agree. Small meals and often are most satisfactory. For the flatulence and borborygmi, alkalis and carminatives are of no assistance, as the gas is lying in a pouch, cut off from the main current of the intestinal tract by the fact that the neck of the diverticulum is kinked with the patient in the upright position.

Rest for half to one hour with the patient horizontal gives good postural drainage. In the writer's case rest in bed was a certain cure. At first sight it would appear useless to consider diet in these cases, as presumably well-mixed chyme leaves the stomach soon after a meal, and in three hours should have entirely passed along. This is so in the healthy intestine, and gas formation—the result of putrefaction or cellulose digestion after a period of hours—has not had time to occur. In the patient with diverticula the food gets into the sac, and, with the neck of the sac kinked, exit is impossible, and with stasis after many hours the sequelæ are obvious.

The non-operative treatment should include:—

1. Rest after meals or at regular intervals during the day—prone or supine.
2. Small light nutritious meals, the ingredients to have as small a residue as possible.
3. Alkalis and bismuth will prevent a too acid and irritating medium coming in contact with the mucosa in the diverticula, already reddened and inflamed with stasis and bacterial infection.
4. Olive oil will act similarly to No. 3.
5. Charkaolin or charecoal in some of its forms will act in conjunction with the above and aid in gas absorption.
6. Abdominal binder or massage may be tried.
7. Gaseous and aerated drinks to be avoided, and fluids to be taken between meals.
8. Straining is to be avoided—urinary straining, straining at stool, heavy physical work, etc. It is noteworthy how many of the patients had enlarged prostate, herniæ, etc.

9. Belladonna or atropine is recommended on the assumption that spasm of the intestine exists which may compress the neck of the diverticula.

10. Intestinal antiseptics, e.g., dimol, etc., if believed in by the physician, may be given.

In the emergency case no clear-cut technique can be followed. Each case is a law unto itself and has to be treated upon its own individual merits.



FIG. 131.—Gut and diverticula filled with bismuth solution.



FIG. 132.—To show diverticula produced by distending jejunum with oxygen.

With perforation, with intestinal obstruction, with fistula formation, and with new growth, the super-added complication must first be treated and the disease proper dealt with later.

In conclusion, only one case in every four needs surgical treatment. The operation of choice is resection of the entire area unless too extensive. If this is impossible, short-circuit, drainage, or plastic operations must be attempted, although not as a rule attended with satisfactory results.

EXPERIMENTAL WORK.

The writer attempted to produce diverticula in the gut by distension alone. The material used was fresh post-mortem gut, and the distending medium was either a thin bismuth solution passed in under pressure by means of a Higginson's syringe, or else oxygen released gradually from a cylinder. With careful and gradual distension diverticula can be produced readily, and



FIG. 133.—Artificially produced diverticula. Note each is related to an artery. Bismuth has been passed through the intestine prior to distension with oxygen. Forceps prevent the gas escaping.



FIG. 134.—Diverticula are small, but their relationship to the mesenteric arteries is noticeable.

from this work the interesting fact resulted that equal distension in the jejunum, ileum, and colon produced diverticula in the jejunum only—which supports clinical facts entirely. Although diverticula are admittedly common in the colon, it is not held by many at the moment that they are the result of distension, which jejunal diverticula undoubtedly are.

The best pictures of these experimental diverticula were obtained by first filling the gut with a thin solution of bismuth and allowing it to run out before the gut was distended with oxygen. This method allows a clear delineation of the mucous membrane and shows that the diverticula produced are in reality herniae of the mucous membrane through the muscle. *Fig. 131* is an example of the artificial diverticula filled with bismuth. *Fig. 132* shows the gut filled with oxygen, while *Figs. 133 and 134* show in addition the fact that these artificial diverticula resemble those found clinically in almost every respect: (1) Most easily produced in the upper jejunum; (2) Always along the mesenteric border and between the leaves of the mesentery; (3) The sacs consist of mucous membrane only; (4) *They are all related to an artery.*

Gradual distension such as the above, no matter how often repeated, always gives the same results. Some workers have tried massive distension of the colon and rectum in living dogs, but this was without result, as one would expect, for it is only jejunal diverticula which are essentially the result of distension, as compared to colonic, which are the result of spasm.

CONCLUSIONS.

An attempt has been made to show the pathology, symptoms, and treatment of that rare condition in which the upper small intestine is studded with multiple false diverticula. The theories regarding the causation and production of such protrusions are considered, and a proposition is formulated, based upon the study of personal clinical material and personal experimental work.

I wish to record my thanks to my chief, Professor Fullerton, for permission to operate upon and publish the case described, which occurred in his wards; for his advice so readily given at all times; and for his kindly criticism which has ever been an incentive and stimulus to me to do better work. I am indebted to Dr. Dixon Boyd for his work in injecting the arteries; to Mr. W. A. C. McConnel for his outline drawings; and to Mr. Leman, of the Radiography Department in the Royal Victoria Hospital, Belfast, for his invaluable assistance so willingly given at all times.

BIBLIOGRAPHY.

- ADAMS, *Brit. Jour. Surg.*, 1926, xiv, 345.
 AKERLUND, A., *Hygiea*, 1919, lxxxi, 449.
 BAASTRUP, CHRISTIAN, *Ugeskr. f. Læger*, 1924, 180.
 BALFOUR, *Ann. of Surg.*, 1923, lvii, 902.
 BICKHAM, *Operative Surgery*, iv, 778.
 BERNER, *Norsk Mag. f. Læge.*, 1906, lxxvii, 1081.
 BERRY, *Lancet*, 1927, ii, 496.
 BETTMAN, R. B., *Surg. Clin. N. Amer.*, 1930, x, 363.
 BÖKER, Dissertation, Kiel, 1912.
 BOLING, J. R., *Jour. Amer. Med. Assoc.*, 1931, xevi, 526; 1930, xev, 267.
 BRAITHWAITE, L. R., *Brit. Jour. Surg.*, 1923, xi, 184.
 BRANDES, K., *Deut. Zeits. f. Chir.*, 1929, ccxv, 390.
 BRISTOW, *Trans. Pathol. Soc. Lond.*, 1854, vi, 191.
 BUCH-HIRSCHFELD, *Lehrb. d. pathol. Anat.*, 1887, ii, 550.
 BUCKWARD and JANICKE, *Deut. Med. Woch.*, 1887, xl, 868.
 BUSCHI, G., *Arch. f. pathol. Anat.*, 1911, cexi, 121.

- BUXTON, ST. J. D., *Jour. of Anat.*, 1923, lviii, 84.
 BUZZI, *Arch. f. pathol. Anat.*, 1885, c, 357.
 BOYD, *Surgical Pathology*, 2nd ed., 355.
 CASE, J. T., *Jour. Amer. Med. Assoc.*, 1920, lxxv, No. 22; *Acta. Radiol.*, 1926, vi, 230;
Jour. Amer. Med. Assoc., 1920, lxxv, 14; 1918, lxx, 1463, 9.
 CHULUMSKY, *Beitr. z. klin. Chir.*, 1899, xv.
 CHRISTIE, *Brit. Med. Jour.*, 1922, i, 990.
 CHRISTOPHER, F., *Internat. Clin.*, 1925, i, 67.
 COLE and ROBERTS, *Surg. Gynecol. and Obst.*, 1920, Oct.
 COOPER, SIR ASTLEY, *Anatomy and Surgical Treatment of Abdominal Hernia*, 1844, 364.
 CORNILLON, *Bull. Soc. Anat. de Paris*,
 EDEL, M., *Arch. f. pathol. Anat.*, 1894, xlv, 400.
 EDWARDS, H., *Brit. Jour. Surg.*, xvii, 7.
 EDWARDS, HAROLD, *Clinical Jour.*, 1928, lvii, 319.
 EVANS, ARTHUR, *Brit. Jour. Surg.*, 1929, xvii, 34.
 EVANS-HOWELL, *Jacksonian Prize Essay*, 1913.
 FORSELL and KEY, *Nord. Med. Arkiv.*, 1915, xlviii, No. 2.
 GIBBERTZ, H., *Arch. f. klin. Chir.*, 1931, clxv, 314.
 GLADTKE, MARTIN, *Deut. Zeits. f. Chir.*, 1930, ccxxiv, 345.
 GODARD, H., *Rev. de Chir.*, 1927, lxx, 22.
 GOOD, Inaugural Dissert., Zurich, 1895.
 GORDINER and SAMPSON, *Jour. Amer. Med. Assoc.*, 1906, xlvi, 1585.
 GRASER, *Arch. f. klin. Chir.*, 1899, lix, No. 3.
 GRASSBERGER, *Wien. klin. Woch.*, 1897, x, 149.
 HANSELMANN, D., *Arch. f. pathol. Anat.* 1896, cxliv, 1.
 HEIDECCKER, *Brunns. Beitr. z. klin. Chir.*, 1928, cxlii, 776.
 HELVESTINE, *Surg. Gynecol. and Obst.*, 1923, July.
 HIRSCHMANN, D., *Jour. Amer. Med. Assoc.*, 1920, No. 22.
 HODENFYL, *Proc. N. Y. Pathol. Soc.*, 1899-1900, 182.
 HUNT, *Surg. Clin. N. Amer.*, v.
 HUNT and COOK, *Ann. of Surg.*, 1921, lxxiv, 746.
 HUNTER, J. T., *Brit. Med. Jour.*, 1922, ii, 800.
 JEFFERSON, G., Personal communication.
 KASPAR, FRITZ, *Arch. f. klin. Chir.*, clxi, 5, 215.
 KEITH, SIR ARTHUR, *Brit. Med. Jour.*, 1920, i.
 KEIBEL and MALL, *Manual of Human Embryology*, 1912, ii, 381.
 LATARGET and MURAD, *Lyon. Chir.*, 1914, xl, 425.
 LEWIS, F. T., and THYNG, F. W., *Amer. Jour. Anat.*, 1907-8, vii, 505.
 LARMORE and GRAHAM, *Surg. Gynecol. and Obst.*, xiv, No. 3.
 MILLER, A. M., *Amer. Jour. Surg.*, 1931, xi, 116.
 MOLESWORTH, H. W. H., *Brit. Jour. Surg.*, 1929, xvii, 342.
 MOLL, *Ibid.*, 1926, xiv, 176.
 MOORE, NORMAN, *Trans. Pathol. Soc. Lond.*, 1884, 202.
 MACKECHNIE, *Ann. of Surg.*, 1921, lxxiii, 96.
 OEHNEL, *Arch. f. Verdauungskr.*, 1923.
 OSLER, W., *Ann. of Anat. and Surg.*, 1881, iv, 202.
 PALMA, MODESTO DALLA, *Pathologica*, 1926, xviii, 65.
 PUGH, see NITCH, C. A. R., *Brit. Jour. Surg.*, 1923-4, xi, 714.
 PIERSOL, *Human Anatomy*.
 REINHARDT, *Zentralb. f. Chir.*, Jahrg 53.
 RANKIN and BROWN, *Surg., Gynecol. and Obst.*, 1930, 836.
 ROEVEKAMP, *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1927, xxxvi, 298.
 ROKITANSKY, *Lehrb. d. pathol. Anat.*, 1856, ii, 36.
 ROTHCHILD, N. S., *Ann. of Surg.*, 1925, lxxvii, 250.
 SHPEPE, W. M., *Jour. Amer. Med. Assoc.*, 1924, lxxxiii, 82, 118.
 SMITHIES, FRANK, *Ibid.*, 1920, No. 22.
 SOMERFORD, A. E., *Lancet*, 1930, i, 1015.
 STEVENSON, H., Personal communication.
 TENGWALL, W., *Acta. Chir. Scand.*, 1931, lxxviii, 162.
 TERRY and MUGLER, *Arch. of Surg.*, 1921, ii, 345.
 TIEDMAN, *Keen's Surgery*, iv, 672.
 TREVES, SIR FREDERICK, *Jacksonian Prize Essay*.
 USLAND, O., *Lecture in Union of Doctors in Trondjhan*.
 VORHANS, M. G., *Jour. Amer. Med. Assoc.*, 1930, xciv, 165.
 WARHASSE, *Surgical Treatment*.
 WATSON, C. M., *Surg. Gynecol. and Obst.*, 1924, xxxviii, No. 1.

A CASE OF LIPOMA OF THE MESENTERY.

BY FRANK D'ABRIEU,

RESIDENT SURGICAL OFFICER, QUEEN'S HOSPITAL, BIRMINGHAM.

Lipomata of the mesentery are classified by Crabtree¹ among the retro-peritoneal fatty tumours. These are usually said to arise from the perirenal fat, but may also originate in fatty tissue in the renal capsule, in the intestinal mesentery, or in the omentum.

Von Wahlendorf reviewed 165 cases of retroperitoneal lipomata and found that 46 per cent of them were pure lipomata and 54 per cent were mixed tumours containing fibrous tissue. The latter are now considered to be sarcomatous in nature and not to be true lipomata.

True lipomata of the mesentery are very rare, and therefore the case recorded below may be of interest. Dannreuther² reports a case where the whole of the mesentery was infiltrated by a diffuse mass of lipomatous tissue in which the abdominal wall was not adipose but showed a remarkable atrophy of the abdominal muscles. Giampaoli³ reported two cases of mesenteric lipomata in 1910 and Altom⁴ another in 1921.

The central areas in a fatty tumour are very liable to necrosis from poor blood-supply. The occurrence of necrosis is said to give rise to fever, chills, and pain, but although the central portion of the tumour was necrotic in this case no febrile symptoms were apparent.

HISTORY.—A. H., a tube-worker, 22 years of age, was sent up to the Queen's Hospital, Birmingham, with a diagnosis of subacute appendicitis. He had been complaining of periodic attacks of pain in the stomach during the previous four months. These attacks, he said, were brought on by certain foods, such as fruit, currant-bread, or parsley sauce. The pain was described as a dull ache in the centre of the abdomen; it was not severe and disappeared after about half an hour. Three weeks before admission he had an attack of severe pain after eating bananas, this time accompanied by nausea which lasted an hour and a half. Two days before admission he received a slight blow in the abdomen from the handle of a gramophone he was winding. This was sufficient to bring on an attack of abdominal pain, which gradually subsided in the evening, but recurred the next day and became gradually more severe, until admission to the Queen's Hospital on the evening of Nov. 1, 1932.

ON EXAMINATION.—The patient was found to be afebrile, but the abdominal muscles were rigid and he was rolling about in bed with pain, making examination difficult. A central abdominal distension was apparent with some visible peristalsis. Two enemata were given, the first producing a poor result and the second none at all. A diagnosis of acute small-intestine obstruction was made.

OPERATION.—An exploration was carried out through a right paramedian sub-umbilical incision. On opening the peritoneum an excess of free fluid was found and a tumour about the size of a large grape-fruit was encountered growing in between the layers of the mesentery of the small intestine. The gut surrounding it was stretched over its surface and closely applied to it anteriorly. The gut in relation to it had undergone a partial torsion and

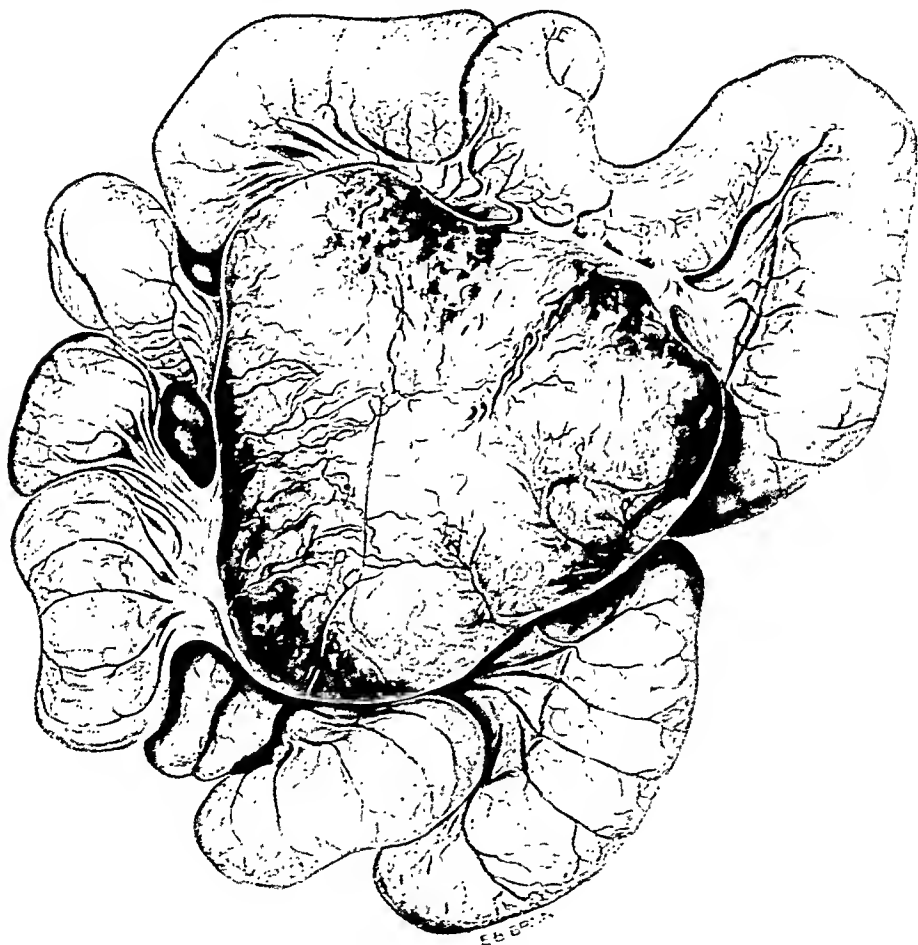


FIG. 135.—Showing the tumour and gut removed at operation.

was bluish in colour but viable. It was feared that attempts at enucleation might damage the blood-supply of an already weakened gut and so a wedge of mesentery containing the tumour, together with about $3\frac{1}{2}$ ft. of gut in relation to it, was excised and an end-to-end anastomosis performed. To prevent undue tension on the line of junction a further side-to-side anastomosis was made between portions of gut above and below, and the abdomen thereafter closed without drainage.

SUBSEQUENT PROGRESS.—The patient made an uninterrupted recovery. His bowels acted without aid the day after operation, and when he began to take a light diet his bowel action was at first (for seven days) a little loose, but this became normal and regular at the end of that time. He left the hospital three weeks after admission, has returned to work, and continued in good health ever since.

PATHOLOGICAL REPORT.—A section from the surface of the tumour, which was preserved in its entirety, was taken and reported on by Professor Haswell Wilson as showing lipomatous tissue with areas of necrosis. To verify the diagnosis of lipoma a deeper section was taken later from the centre of the tumour, and was reported on on Dec. 17 as follows: "Sections from the deeper part of the growth showed the same appearances as those formerly described, viz., a lipoma with areas of necrosis".

The accompanying illustration (*Fig. 135*) shows the tumour and gut removed at operation. There are whitish areas around the circumference of the lipoma which were due to rupture and diffusion of the contents of the lacteal vessels and presented a remarkable picture at operation. The lower part of the tumour shows an area where necrosis had reached the surface.

I wish to thank Mr. Sankey, at whose direction I operated on this case, for permission to report it.

REFERENCES.

- ¹ CRANTHILL, E. G., "Pararenal Lipoma with Generalized Abdominal Lipomatosis", *Jour. of Urol.*, 1930, xxiii, 545.
- ² DANNREUTHER, W. T., "Mesenteric Lipomatosis and Megacolon, with Muscular Atrophy of Abdominal Wall: Report of Case", *Amer. Jour. Obst. and Gynecol.*, 1928, xvi, 267.
- ³ GIAMPAOLA, A., "Cirea due Casi di Lipoma del Mesentere", *Policlinico (Sec. prat.)*, 1910, xvii, 370.
- ⁴ ALTON, B. H., "Lipoma of the Mesentery", *Boston Med. and Surg. Jour.*, 1921, clxxv, 205.

OCCUPATIONAL ANEURYSM OF THE PALMAR ARTERIES.*

BY D. STEWART MIDDLETON,

HON. SURGEON, THE CHURCH OF SCOTLAND DEACONESS HOSPITAL; HON. ASSISTANT SURGEON,
THE EDINBURGH ROYAL INFIRMARY.

A SURVEY of the literature of aneurysms of the palmar arteries yields records of 70 indubitable cases. Some references of older authors could not be traced, and some cases have been discarded on account of insufficiency of detail.

Like aneurysms elsewhere, those of the palmar arteries may be divided into false, of which there were 54 cases, and true aneurysms, of which 16 require consideration. The false aneurysms, usually referred to by the too inclusive and therefore somewhat misleading term 'traumatic aneurysm', resulted from punctured wounds of the hand, with a solitary exception when the aneurysm followed the subcutaneous rupture of an artery during reduction of a metacarpophalangeal dislocation of the thumb. This type of palmar aneurysm, following transfixion or laceration, does not come further within the purview of the present communication.

True aneurysm, a single, localized ectasia of one of the palmar arteries, is of relatively rare occurrence, and the majority of such cases seem to be traumatic in origin. A single case is recorded¹⁸ in which the arterial affection, associated with subacute endocarditis, was considered to have followed the blocking of the vessel by an embolus. The likelihood of such an occurrence seems doubtful.

Where no obvious cause can be assigned, aneurysm of the palmar arteries has been termed 'spontaneous'. Only two cases of this variety are forthcoming, that of Griffiths,³ an aneurysm of the ulnar artery in a young woman, and that of Reid,¹⁵ where the aneurysm was not palmar but occurred on the back of the hand in a tobacco-dealer. Apart from these cases in which investigation failed to find adequate explanation for the aneurysm there are those which more nearly concern this paper, being traumatic in origin, though not dependent on an open wound. There are two varieties, both occupational, one acute and due to a single definite contusion, the other of more delayed and gradual development, the result of repeated minor injury.

Of the acute traumatic aneurysm of the palmar arteries, eight cases are recorded,^{6, 10, 13, 14, 16, 17, 19} of which the following three may be abbreviated as types:—

W. Robertson¹⁶ in 1897 described the case of a railway fireman at Perth, aged 18, who dealt the 'injector' of his engine a blow with the palm of his left hand, suffering considerable pain as a result. An aneurysm, probably of the ulnar artery in the hypothenar region, was noticed five months later.

* From the Department of Surgery, University of Edinburgh.

D. B. Robinson¹⁷ places on record the case, in Kansas City, of a railway switchman who had also been a prizefighter. He struck the tap of an air-hose with the palm of the right hand and felt severe pain radiate to the fingers. One month later he came under treatment for an aneurysm of the deep palmar arch.

Regnault and Bourrut-Laconture¹⁴ published the case of a second master gunner in the French navy, aged 37 years. During firing practice the breech of his gun jammed and he dealt it several hard blows with the palm of his left hand in order to free it. He felt pain in the hypothenar region at the time, and three weeks later he was admitted to a naval hospital with an aneurysm of the hypothenar extremity of the superficial palmar arch.

The more strictly occupational palmar aneurysms, with which this paper is particularly concerned, are of that type which succeed prolonged and frequently recurring minor traumata, the trauma being, as a rule, incidental to the individual's work. This type best merits the term 'occupational' aneurysm, and of it only six cases are on record. Of these a short individual summary is of interest :—

Guattani,¹—In 1772 there was published what appears to be the earliest fully recorded case of aneurysm of the ulnar artery in the palm. The side affected is not specified. More recent writers refer to the affected individual as 'coachman'⁸ and attribute the aneurysm to the recurring trauma of handling reins and whip. Setting aside the fact that in 1772 throughout Continental Europe it was universal to ride postilion and not drive from the box, a study of Erichsen's classic translation from the original Latin of Guattani's paper² makes this man's real occupation clear. The passage in question runs as follows: "A groom, Johannes Perisius by name, a Roman, 45 years of age, of a sanguineous temperament, a great wine drinker and now the horse-keeper of the most excellent Marquis Angelo Gabrielli." Neither here nor elsewhere in the translation is there any mention of reins or whip, and the author does not draw attention to any chronic trauma to which the man was exposed. It appears reasonable, however, to suppose that a Roman groom in 1772 would be exposed to a considerable amount of arduous manual toil, and the case is therefore included here among the group of 'occupational' aneurysms.

Morestin,¹²—A carter, aged 43, whose work entailed the handling of many large and heavy boxes, developed a left ulnar aneurysm in the palm. The author concludes that the aneurysm is attributable to the man's occupation combined with a possible arterial degeneration due to an alcoholic disposition.

Duvernoy,¹—A blacksmith, aged 50, developed an aneurysm of the thenar extremity of the right superficial palmar arch attributed to the repeated trauma of swinging a heavy hammer.

Maclaure,¹¹—A joiner acquired an aneurysm of the right superficial palmar arch as a result of the frequent use of a hand-plane.

Lejars,⁷—A book-carrier, aged 35, developed an aneurysm in the hypothenar region, caused apparently by the trauma of the edges and corners of the piles of books which he carried for a livelihood.

Marques and Marques,⁹—An aneurysm of the right deep palmar arch resulted from the recurring trauma of the implements used by an agricultural labourer.

To these records I am enabled to contribute the two following cases :—

Case 1.—A male, aged 38 years, attended the Surgical Out-patient Department of the Edinburgh Royal Infirmary towards the end of 1932. He was a healthy man in all respects save for the loss of his right leg during the war. His Wassermann blood-reaction was negative.

During ten and a half years he had earned a living as a wood-machinist. Most of his work was concerned with cutting six-inch lengths off long planks of wood. For this purpose he used a mechanical saw, and before cutting the wood he pushed the plank into position by a blow with the hypothenar region of his right hand. Furthermore, he had formed the habit, since the loss of his leg, of raising himself from the sitting position by pressing the palms of his hands against the edge or corners of the chair.

For some months past, while performing these movements, he had felt as if something deep in the hypothenar eminence slipped over an underlying bone. When this occurred pain was referred to the hypothenar eminence at a point about a finger-breadth distal to the pisiform bone. The pain did not radiate to the fingers, nor had he ever experienced paræsthesia referable to the ulnar-nerve distribution. There was no sign of ulnar-nerve weakness, but on careful palpation a small hard mass about the size of a pea could be felt to slip over the hook of the hamate (unciform) bone, and this movement reproduced the characteristic pain complained of.

At operation an incision was made in the line of the ulnar artery and nerve. These structures, where they entered the palm, were followed distally. Over the hook of the hamate bone the ulnar artery, which was more tortuous than normally, formed an aneurysmal swelling, which was filled with firm coagulum. The clot extended distally a short distance beyond the aneurysmal sac, and ceased abruptly where the artery divided into superficial and deep branches. The artery proximally and the superficial and deep branches distally were ligated. The intervening segment was then removed (*Fig. 136*).

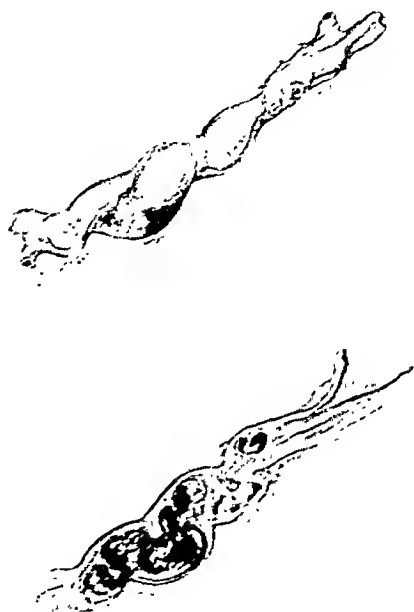


FIG. 136.—*Case 1.* Aneurysm of the ulnar artery in the palm.

Case 2.—I am indebted to Mr. D. M. Greig, Conservator of the Museum of the Royal College of Surgeons of Edinburgh, for permission to record an additional experience.

In 1888, while serving in India, Mr. Greig, as Orderly Officer in the Mian Mir cantonment of Lahore, was called to hospital to a private of the 33rd West Riding of Yorkshire Regiment who was bleeding from the right hand. The soldier was of more than seven years' service, and except for malaria had a clear medical history sheet; he denied syphilitic infection, and nothing to suggest syphilis could be found on general examination.

He had been admitted to hospital on account of a swelling in the right

thebar region. Diagnosed as an abscess, incision was followed by profuse hæmorrhage, which was controlled by packing. The hæmorrhage had recurred on more than one occasion and the tampon had been each time renewed. Mr. Greig ligated the superficial volar branch of the radial artery above and below an aneurysmal sac, and later the man returned to duty.

Investigation yielded a clear statement of the causal trauma. In firing his rifle he did not follow the drill-book instructions and brace the butt firmly against his body. He habitually helped to check the recoil by the strong grasp of his hands. He was always conscious of the recoil, not merely against his shoulder, but also against the base of his right thenar eminence on the 'grip' of the rifle. The military rifle in use in India at that time was the Martini-Henry. To replace each cartridge the breech was opened by depressing with the right hand a lever which lay close to and behind the trigger-guard. The action of sharply pushing down the lever was performed by the thumb, and the ball of the right thumb came abruptly in contact with the lever as it reached its limit of movement. Thus, recurring trauma to the superficial volar branch of the radial artery at the base of the thenar eminence was the main etiological factor in the production of an aneurysm.

SUMMARY.

1. Aneurysms of the palmar arteries are divided into: (a) False aneurysms; and (b) True aneurysms, which are frequently of occupational origin. The previously recorded cases of the latter are surveyed.

2. Two hitherto unpublished cases of true occupational aneurysms in the palm are placed on record.

REFERENCES.

- ¹ DUVERNOY, *Thèse de Paris*, 1870 (quoted by Lyle⁸ and Volkmann¹⁹).
- ² ERICHSEN, J. E., *Observations on Aneurysm*, 1844, 268. London: Sydenham Society.
- ³ GRIFFITHS, J., *Brit. Med. Jour.*, 1897, ii, 646.
- ⁴ GUATTANI, C., *De externis aneurysmatibus manu chirurgica methodice pertractandis*, 1772. Rome. (Translated by Erichsen.²)
- ⁵ KEEN, W. W., *Int. Clin.*, Philadelphia, 1893, 3rd s., ii, 194.
- ⁶ KENDRIZY and RECLUS, *Thèse de Paris*, 1914, No. 159 (quoted by Lyle⁸).
- ⁷ LEJARS, F., *Semaine méd.*, 1914, xxxiv, 328.
- ⁸ LYLE, H. H. M., *Trans. Amer. Surg. Assoc.*, 1924, xlii, 573.
- ⁹ MARQUES and MARQUES, *Ann. Paulist. Med. Cir.*, 1920, xi, 159 (quoted by Lyle⁸).
- ¹⁰ MATAS, reported by Lyle.⁸
- ¹¹ MAUCLAIRE, *Bull. et Mém. Soc. Chir. de Paris*, 1912, xxxviii, 466.
- ¹² MORESTIN, H., *Bull. et Mém. Soc. Anat. de Paris*, 1905, lxxx, 556.
- ¹³ RASTOUIL (rapport de Robineau), *Bull. et Mém. de Soc. Chir.*, 1913, xxxix, 15.
- ¹⁴ REGNAULT, J., and BOURRUT-LACOUTURE, *Rev. de Chir.*, 1913, xlvii, 337.
- ¹⁵ REID, M. R., *Arch. of Surg.*, 1926, xii, 1.
- ¹⁶ ROBERTSON, W., *Brit. Med. Jour.*, 1897, ii, 1637.
- ¹⁷ ROBINSON, D. B., *Jour. Amer. Med. Assoc.*, 1912, lviii, 32.
- ¹⁸ SOUPAULT, R., GUTMAN, R. A., ROUCHÉ, P., and JOHIEL, R., *Presse méd.*, 1929, xxxvii, 236.
- ¹⁹ VOLKMANN, J., *Deut. Zeits. f. Chir.*, 1930, cccxxvii, 151.

COCCYGEAL SINUS.

BY R. L. NEWELL, MANCHESTER.

I HAD some difficulty in choosing a title for this paper. Although coccygeal sinus or fistula is the name by which the condition is known in English literature, it is an inaccurate description because the only connection the sinus has with the coccyx is solely one of position. 'Congenital sinuses in the sacrococcygeal region' is perhaps a better title. In America Hodges²¹ as long ago as 1880 manufactured the name of 'pilonidal sinus', or nest of hairs.

The condition has received very little attention by British surgeons. Goodsall and Miles¹ in their book on *Diseases of the Anus and Rectum* devote a whole chapter to a condition which they call 'sinus over the sacrum or coccyx', but they fail to recognize its true nature, and attribute its cause to injury. They recognize, however, that it has no connection with the coccyx or sacrum. Even as late as 1932 W. E. Miles²¹ still regards it as due to a hæmatoma resulting from a fall, and advances the theory that right-handed people fall more heavily on the left side, accounting for the fact that these sinuses appear to originate on the left side of the median raphe.

Lockhart Mummery² drew attention to the congenital origin of the condition in 1921 and demonstrated that the sinuses were lined by epithelium. and later in 1929 published an excellent microphotograph in the *Proceedings of the Royal Society of Medicine*.³ Towards the end of 1932 W. B. Gabriel²¹ published his book *The Principles and Practice of Rectal Surgery*; in it he described the results of his treatment in 9 cases.

It is a condition, however, which has received considerably more attention in America. Harvey B. Stone⁴ reports 61 collected cases. P. B. Cattell and L. W. Stoller⁶ report 59 cases from the Lahey Clinic. and Frank Glenn²³ reports 120 cases. These figures give the impression that its occurrence is far commoner in America than it is in this country. Personally I am of the opinion that this is not the case, and that the lack of more frequent reference to it in our surgical literature is due to the failure on the part of the practitioner or surgeon to appreciate the pathological basis of the condition. Failure to understand the true nature of the sinus may lead to the mistaken diagnosis of fistula in ano, tuberculous sinus, or even a simple abscess or boil.

I do not doubt that there is hardly a surgeon in this country who has not seen or operated upon several of these cases. and possibly been disappointed with the result of his surgical intervention. P. B. Cattell and L. W. Stoller⁶ have followed up 40 cases, and in 9 have found recurrences: 20 out of 50 of their cases had had previous operations.

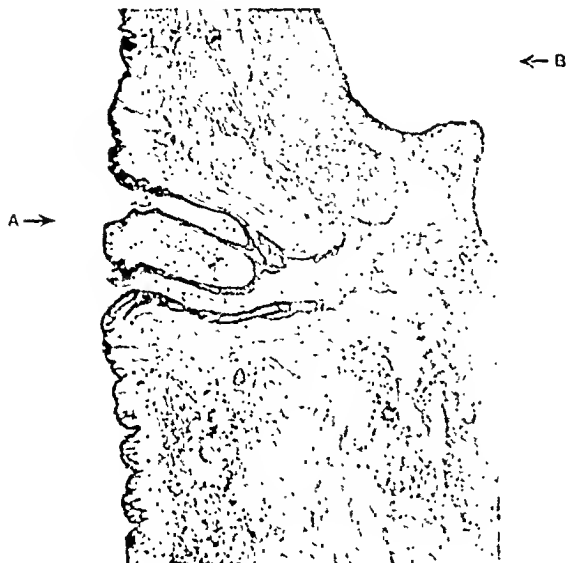


FIG. 141.—A section of specimen shown in Fig. 147 taken in the mesial plane. It shows two primary sinuses in the median raphe, one above the other. These two sinuses join and extend backwards towards the coccyx. The epithelium appears to be intact. The secondary sinus was caused by one of the other two primary sinuses present in this case. A, Two small primary sinuses in the median raphe; B, Secondary sinus.

FIG. 142.—A vertical section in the coronal plane at right angles to Fig. 140.

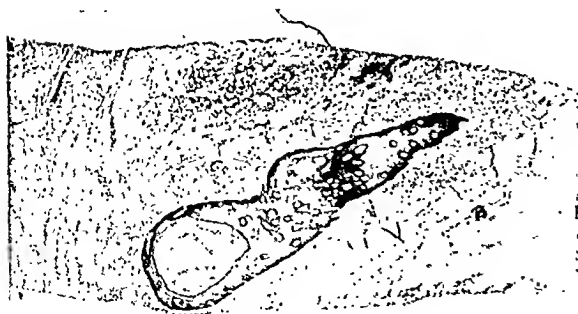


FIG. 143.—A vertical section nearer the coccyx than Fig. 142. The sinus is now expanding into a small cavity.

by the microphotographs (*Figs. 140-144*). The sinus B (*Fig. 137*) is purely a secondary sinus and is lined only by granulation tissue. This accounts for the fact that the pathologist, on examining the tissue removed, will often send back a report that it is septic granulation tissue.

ETIOLOGY.

The exact etiology of the condition has not yet been proved conclusively, but it probably results from change in embryonic development. Harvey B. Stone⁴ gives to J. M. Warren⁷ the credit of first describing the condition in 1867. Warren regarded a reversed hair follicle as being the cause of the trouble, and as the hair continues to grow "it pulls its hole in after it."

Wendelstadt⁸ advances the following theory. The inferior end of the spinal cord is the last portion to lose its connection with the skin. As the sacrum closes together it pinches off this connection, and the skin becomes attached by fibrous tissue at this point to the posterior surface of the sacrum. As adult life approaches, fat and soft parts grow and lift the skin farther away from the bone, and the attached spot is drawn down into a dimple or sinus. P. B. Cattell and L. W. Stoller⁶ give Oehlecker's⁹ views without comment. He believes that these sinuses are caused by the traction of the caudal ligament on the median raphé. Hermann and Tournaux²² regarded the condition as due to a vestigial remnant of the spinal cord. They state that after the separation of the spinal cord from the superficial tissues a process of epithelial tubules persists for a time at the point of previous connection of the neural axis with the skin, forming a vestigial remnant of that connection.

Harvey B. Stone,⁵ in a more recent paper, suggests as a possible cause that the sinuses are analogous to the preen glands found in a great many species of birds. The preen gland lies embedded in fat over the last caudal vertebra. It consists of numerous straight tubules lined by polyhedral cells. The tubules converge upon a collecting chamber or cavity, which in turn empties into an epithelial-lined duct opening on to the skin of the back. Their function is either that of oiling feathers or it is sexual.

Ripley¹⁰ and Moise¹¹ both regard the condition as analogous to spina bifida, and have recorded two cases of staphylococcal meningitis resulting from a congenital sacral sinus. In both their cases there was, however, a direct communication with the spinal meninges and a maldevelopment of the vertebral bodies. Bland-Sutton¹² considers that the condition is due to faulty coalescence of the cutaneous covering of the back, and mentions as analogous the interdigital pouch of the sheep, which often gives trouble if



FIG. 144. — High-power view of *Fig. 143*, showing a cavity containing hairs and epithelial debris and lined by epithelium.

the orifice becomes occluded. He says that they are sinuses frequently associated with lumbosacral spina bifida. This statement, however, is not corroborated by an analysis of the published cases.

It would appear, then, that we have to consider two main theories:—

1. That the condition is a true sequestration dermoid, due to faulty coalescence of the middle line.

2. That the condition arises from that remnant of the spinal canal known as the coccygeal vestige.

I do not think the condition is entirely analogous to a true sequestration dermoid. Coccygeal fistulae are comparatively frequent, and are in a constant position, whereas dermoid fistulae arising in the middle line are very rare in other parts of the body. I therefore consider that the area in which they are found has a definite influence on their etiology.

As regards the theory that they arise from the coccygeal vestige, we have the work of Hermann and Tournaux²² to support this hypothesis. They showed that in the fetus at a definite stage there is a connection of the coccygeal vestige with the skin. On the other hand, recent work by Kunitomo,²⁸ who had available for his research the unrivalled collection of embryos at the Carnegie Institute, has failed to confirm this statement. Kunitomo states, "The caudal end of the coccygeal medullary vestige appears to adhere to the epidermis, but in reality it does not."

What, however, is very significant in his work is that he found the various stages in the reduction of the tail bud, as shown on the skin, do not present the same appearance in every embryo, but, on section, evidence of its reduction and disappearance are invariably found dorsal to the caudal end of the vertebral column and in the median line of the embryo.

A coccygeal fistula is lined by tissue indistinguishable from true skin. If it arose from the coccygeal vestige, the cells lining it would have become so differentiated as to be quite unlike true skin. In microscopic sections of the condition there is absolutely no sign of any other tissue than that normally contained in the skin.

I venture to suggest that the following is the true origin of the condition. The sinus is a dermoid fistula, but differing from a true sequestration dermoid in that it is caused by traction of the underlying tissue upon the median raphe. This traction is probably caused by the retrogression of the tail bud. It therefore could be described as a traction 'dermoid'. It may be suggested that the following is the probable sequence of events. The epithelial-lined fistula is present at birth. It would appear to remain a considerable time without giving rise to symptoms. However, owing to the growth of the body and consequent enlargement of the buttocks, the secretion of this epithelium is unable to escape by the orifices in the middle line. These orifices are in a position which is extremely liable to sepsis, being situated at the bottom of a fairly deep cleft. The epithelial fistula becomes infected, and the formation of an abscess follows. This abscess disintegrates a portion of the epithelial lining and a sinus track is formed which tends to extend along the line of least resistance, which is upwards and outwards. This secondary sinus eventually opens on to the surface to one side of the middle line.

CASE REPORTS.

The following cases have been operated upon by me by the method I am about to describe. In all cases the condition appears to have been completely cured.

Case 1.—M. C., female, aged 26. The sinus had been discharging for twelve months. There were three primary sinuses in the middle line and one secondary sinus opening on the left of the middle line. Operation on Feb. 5, 1932. The wound became septic and healed by granulation tissue.

Case 2.—C. J., female, aged 30. The sinus had been discharging for two years. She gave a history of one previous operation. There were two primary sinuses in the middle line and one secondary sinus opening on the left of the middle line. Operation on Feb. 10, 1931. Wound healed by first intention.

Case 3.—G. A., female, aged 37. The sinus had been discharging for two years. She gave a history of one previous operation. There were three primary sinuses in the middle line and one secondary sinus opening on the right of the middle line. Operation on July 26, 1932. The wound became septic and healed by granulation tissue.

Case 4.—M. P., female, aged 20. The sinus had been discharging for two and a half years. She gave a history of one previous operation. There was one primary sinus only in the middle line. Operation on Sept. 29, 1932. The wound healed by first intention.

Case 5.—E. H., female, aged 25. The sinus had been discharging for ten years. She gave a history of three previous operations. There were two primary sinuses in the middle line and one secondary sinus opening on the left of the middle line. Operation on May 31, 1932. The wound became septic and healed by granulation tissue.

Case 6.—H. P., female, aged 25. The sinus had been discharging for four years. She gave a history of one previous operation. There were two primary sinuses in the middle line and one secondary sinus on the left of the middle line. Operation on Nov. 4, 1931. The wound became septic and healed by granulation tissue.

Case 7.—F. H., female, aged 30. The sinus had been discharging for six months. There was one primary sinus in the middle line and one secondary sinus to the left of the middle line. Operation on Feb. 8, 1933. The wound became septic and healed by granulation tissue.

Case 8.—E. W., female, aged 32. The sinus had been discharging for twelve years. She gave a history of two previous operations. There were four primary sinuses in the middle line and one secondary sinus on the left of the middle line. Operation on Feb. 10, 1933. The wound became septic and healed by granulation tissue.

Case 9.—K. B., female, aged 23. The sinus had been discharging for two and a half years. She gave a history of several incisions. There were four primary sinuses in the middle line and one secondary sinus on the left of the middle line. Operation on Feb. 14, 1933. The wound became septic and healed by granulation tissue.

Case 10.—M. B., female, aged 23. The sinus had been discharging for one year. She gave a history of several incisions. There were two primary sinuses in the middle line and one secondary sinus on the left of the middle line. Operation on Jan. 31, 1928. The wound became septic and healed by granulation tissue.

Case 11.—A. S., female, aged 20. The sinus had been discharging for two years. She gave a history of two previous incisions. There was one primary sinus in the middle line and evidence of a healed secondary sinus to the left of the middle line. Operation on March 17, 1933. The wound became septic and is healing by granulation tissue at the time of writing.

All these cases occurred in women. Frank Glenn's figures show, however, that it occurs more frequently in men. There were 23 females and 97 males in his 120 collected cases. The earliest onset of symptoms was at the age of 15 years, and the latest onset at the age of 35 years.

All these cases were septic at the time of operation, and primary healing was obtained in only two of the series. The remainder healed by granulation tissue. The time taken for complete healing varied from three to ten weeks. In no case have the symptoms recurred.

TREATMENT.

Although successful treatment has been reported with non-surgical methods by Maillard¹² and Crookall,¹¹ it would appear that the only satisfactory way of curing the condition permanently is by surgical excision. A complete radical excision which removes the whole of the area involved is absolutely necessary, otherwise recurrence is inevitable. Failure to undertake a preliminary investigation of these cases with a view to obtaining a true diagnosis and an accurate delimitation of the sinus results in many of the patients having to undergo several operations before obtaining a cure. The following method has been found to give very satisfactory results:—

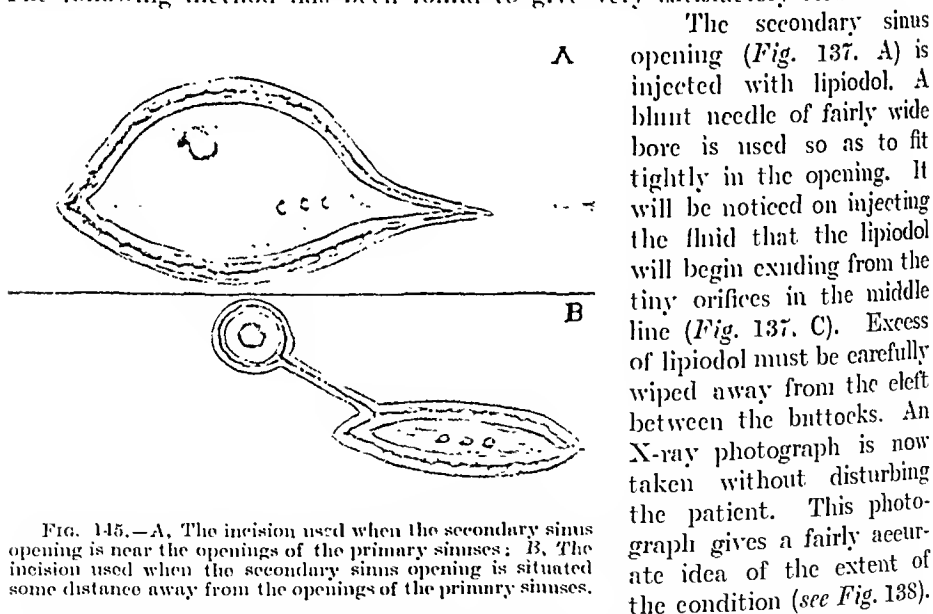


FIG. 145.—A, The incision used when the secondary sinus opening is near the openings of the primary sinuses; B, The incision used when the secondary sinus opening is situated some distance away from the openings of the primary sinuses.

The secondary sinus opening (*Fig. 137. A*) is injected with lipiodol. A blunt needle of fairly wide bore is used so as to fit tightly in the opening. It will be noticed on injecting the fluid that the lipiodol will begin exuding from the tiny orifices in the middle line (*Fig. 137. C*). Excess of lipiodol must be carefully wiped away from the cleft between the buttocks. An X-ray photograph is now taken without disturbing the patient. This photograph gives a fairly accurate idea of the extent of the condition (*see Fig. 138*).

Immediately before the operation, when the patient is under the anæsthetic, the opening of the secondary sinus (*Fig. 137. A*) is injected with melted paraffin wax. Coloured wax can be employed, but there is no advantage in this, as the wax is only used to make the whole of the sinus palpable and to serve as an indication if the sinus is cut across during the operation. A circular incision is now made around the orifice of the secondary sinus, and a straight incision directly over the underlying secondary sinus is made downwards towards the middle line (*Fig. 145B*). That portion of the median

raphé which is affected is now excised by an elliptical incision. If the secondary sinus opening lies near the middle line, then the incision must include both primary and secondary openings (*Fig. 145A*). The incision in *Fig. 145B* will, however, facilitate the closure of the wound. The skin-flaps are dissected well back, taking care to keep fairly superficial (*Fig. 146*). The whole of the sinus track is now removed (*Fig. 147*), beginning at the upper portion. There is no need to make a wide excision of the track from A to B (*Fig. 137*), as the track in this situation is lined only by granulation tissue and has no epithelium lining its walls. The condition, however, is different when the middle line is approached. Here we are dealing with the primary sinus, which has an epithelial lining, and it is advisable to excise not only the sinus but also the skin of the median raphé

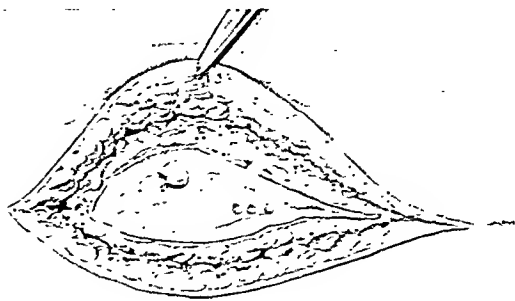


Fig. 146.—The skin edge is undermined.



Fig. 147.—Case 8. Photograph of the sinus removed. The lower portion of skin shows four small openings in the median raphé. The narrow portion of skin passing upwards and to the left is overlying the secondary sinus. The opening of the secondary sinus can be seen at the top of the specimen.

and a generous portion of tissue around the sinus. Otherwise it is possible that any pits or depressions of the epithelium lining may be cut across and left behind.

This dissection will, of course, leave a fairly large cavity extending forwards to the coccyx, and an attempt must be made to obliterate this cavity as far as possible by undermining the fat and approximating it with catgut sutures. The skin is then sutured without drainage. An application of new skin helps to prevent contamination of the wound. If the wound becomes septic, as is not improbable, then the sutures must be removed and the wound allowed to heal by granulation tissue. Irrigation with a eusol solution should be performed twice daily, and later light packing with an allantoin solution will be found very efficacious in stimulating granulation tissue formation. Every effort should be made to obtain primary healing by swabbing the cavity with B.I.P.P., by undermining the fat, and by obliteration of the cavity with as little tension as possible by means

of catgut sutures through the fat. If primary healing is not obtained, it may be as long as two months before healing by granulation tissue takes place.

CONCLUSIONS.

1. A coccygeal sinus must be regarded as due to a defect in embryonic development. It probably is the result of traction on the skin caused by retrogression of the tail bud.

2. The treatment consists in the removal of the whole of the sinus, together with that portion of the median raphe which contains the origin of the condition.

3. The extent of the sinus may be difficult to recognize without a lipiodol injection, followed by an X-ray examination.

4. The dissection is rendered easier by an injection of paraffin wax immediately prior to operation.

I wish to tender my thanks to Mr. John Morley and Professor J. S. B. Stopford for their helpful advice in the preparation of this paper, and to Dr. G. E. Loveday, and Dr. E. R. A. Cooper for the preparation of the microscopic sections.

REFERENCES.

- ¹ GOOSALL, D. H., and MILES, W. E., *Diseases of Anus and Rectum*, 1900.
- ² LOCKHART-MUMMERY, J. P., *Brit. Med. Jour.*, 1921, i, 807.
- ³ LOCKHART-MUMMERY, J. P., *Proc. Roy. Soc. Med.*, 1920, xxii, 1331.
- ⁴ STONE, H. B., *Ann. of Surg.*, 1924, lxxix, 410.
- ⁵ STONE, H. B., *Ibid.*, 1931, xciv, 317.
- ⁶ CATTILL, P. B., and STOLLER, L. W., *New Eng. Jour. of Med.*, 1932, ccvi, 110.
- ⁷ WARREN, J. M., *Surgical Observations*, 1867, 192. Boston. (Quoted by Stone.⁴)
- ⁸ WENDELSTADT, H., *Ueber Hauteinwachsungen und haarhaltige Fisteln in der sacrococcygeal Gegend*, 1885. Bonn. (Quoted by Stone.⁴)
- ⁹ OEBLECKER, F., *Deut. Zeits. f. Chir.*, 1926, exxvii, 262. (Quoted by Cattell and Stoller.⁶)
- ¹⁰ RIPLEY, F. H., *Amer. Jour. Dis. Child.*, 1928, xxxvi, 785.
- ¹¹ MOISE, T. S., *Surg. Gynecol. and Obst.*, 1926, xlii, 394.
- ¹² BLAND-SUTTON, J., *Tumours*, 1922, 7th ed.
- ¹³ MAILLARD, E. R., *Jour. Amer. Med. Assoc.*, 1929, xciii, 1383.
- ¹⁴ CROOKALL, A., *Trans. Amer. Proctol. Soc.*, 1928, xxviii, 32.
- ¹⁵ LAUEY, F. H., *Surg. Gynecol. and Obst.*, 1929, xlviii, 109.
- ¹⁶ LAUEY, F. H., *Ibid.*, 1932, liv, 521.
- ¹⁷ DULLIGAN, A. J., *Amer. Jour. Surg.*, 1929, vi, 554.
- ¹⁸ BHAMS, J., *Radiology*, 1927, viii, 248.
- ¹⁹ HARDMAN, W., *Lancet*, 1882, i, 501.
- ²⁰ MILES, W. E., *Brit. Med. Jour.*, 1921, i, 915.
- ²¹ MILES, W. E., *Proc. Roy. Soc. Med.*, 1932, xxv, 1649.
- ²² TOURNEAUX, F., and HEHMANN, G., *Jour. Anat. et Physiol.*, 1887, xxiii, 498.
- ²³ GLENN, FRANK, *New Eng. Jour. of Med.*, 1932, ccvii, 544.
- ²⁴ GABRIEL, W. B., *The Principles and Practice of Rectal Surgery*, 1932.
- ²⁵ NORBURY, L. E. C., and MORGAN, C. N., *Proc. Roy. Soc. Med.*, 1927, xx, 1274.
- ²⁶ ARRY, L. B., *Developmental Anatomy*, 1932, 2nd ed.
- ²⁷ ALEGAIS and PEYRON, *Compt. rend. Soc. Biol.*, 1920, lxxxiii, 230.
- ²⁸ KUNITOMO, K., *Carnegie Contributions Embryol.*, 1918, viii, No. 26, 167.
- ²⁹ ILLINGWORTH, C. F. W., and DICK, B. M., *Surgical Pathology*, 1932.
- ³⁰ GANT, S. G., *Diseases of the Rectum, Anus and Colon*, 1923, i.
- ³¹ HODGES, *Boston Med. and Surg. Jour.*, 1880, ciii, 485, 493 and 544. (Quoted by Stone.⁴)

ASSOCIATED FACIAL AND INTRACRANIAL HÆMANGIOMATA.*

By LAMBERT ROGERS,

ASSISTANT DIRECTOR OF THE SURGICAL UNIT, WELSH NATIONAL SCHOOL OF MEDICINE,
THE ROYAL INFIRMARY, CARDIFF.

THE different forms to which anomalous development of angioblastic tissue may give rise in connection with the central nervous system and its envelopes are gradually being recognized. The presence of such lesions, though rare, can in some instances be suspected, but the question—at times a difficult one—of their most satisfactory treatment is still under consideration.

The following case exemplifies an extensive and somewhat rare distribution of hæmangiomatous tissue in connection with the fore-brain and its coverings.

CASE REPORT.

On Dec. 3, 1931, a well-built boy, aged 8, was admitted to the Surgical Unit on the recommendation of Dr. Ivor Davies, who had seen him in his out-patient department. The child's mother stated that he had been subject to fits for about seven years, and that the fits occurred at intervals of approximately three weeks. Each fit was characterized by loss of consciousness, lasted for about two or three minutes, and was followed by "paralysis of his left side", which sometimes remained as long as three days but then passed off.

ON EXAMINATION.—Examination revealed an extensive 'port-wine' stain over the right side of the face and forehead (*Fig. 148*), brisk knee-jerks, the left a little brisker and more pronounced than the right, and flexor plantar reflexes. Nothing else abnormal was noted.

A report on the fundi was made by Dr. Leighton Davies, as follows: "There is no optic neuritis in either eye, but the left disc appears a little abnormal. It is almost impossible to get more than a fleeting glimpse, but apparently the centre of the disc is very pale. I think this is probably due to an abnormally deep and large physiological cup. ? one small hæmorrhage in left retina. No engorgement of vessels."



FIG. 148.—Boy, aged 8, with fronto-parietal extra- and intradural plexiform angiomata on the same side as an extensive capillary nævus, visible in the photograph.

* From the Surgical Unit, The Welsh National School of Medicine, The Royal Infirmary, Cardiff.

While the patient was in hospital the fits became more frequent; three occurred over one week-end. A characteristic fit is described as follows: "There was no cry, the arms and legs were thrown about, and all four limbs twitched violently. Breathing was laboured and rapid." After certain, but not all, fits there was a left hemiparesis.

The skull was X-rayed by Dr. Owen Rhys, and plates showed an opacity in the right fronto-parietal region (*Figs. 149, 150*).

DIAGNOSIS.—Owing to the increasing frequency of the fits followed by transient attacks of left hemiplegia, and the X-ray evidence of an intracranial opacity, it was decided to carry out an exploratory osteoplastic craniotomy over the right frontal lobe. A tentative diagnosis of a partly calcified hæmangioma was made, because of the attacks of convulsions followed by fleeting hemiplegia and the presence of the facial naevus.

FIRST OPERATION.—On Jan. 25, 1932, under intrapharyngeal chloroform and ether anaesthesia, an osteoplastic flap was cut with the skull plough. The flap was designed to expose the outer aspect of the right frontal lobe and to have its base beneath the temporal muscle. The bone-flap was turned down and the exposed dura mater was seen to be tense and not pulsating. The middle meningeal vessels passed upwards and backwards across it, and in the middle of the field an intricate anastomosis of fine branches lay across the main meningeal vessels, forming a plexiform angioma about the size of a large almond (*Fig. 151*).

The meningeal vessels were ligated with fine catgut as near their origin as possible. The dura was opened well away from the edges of the blood-vessel tumour, some outlying branches of the

meningeal vessels being divided between silver clips. The frontal lobe bulged slightly through the wound as the dura was divided, and the whole surface of the exposed cortex was found to be beset with fine anastomosing vessels. The dura mater was anchored to the brain by a leash of vessels which passed from the deep surface of the extradural hæmangioma to the face of the similar but more extensive cortical tumour. Silver clips were placed on the vascular leash, which was afterwards divided between them so that the dural flap could then be thrown backwards on its base (*Fig. 152*). The brain was now seen to be pulsating normally. A small subtemporal bone defect was produced for decompression at the base of the bone-flap, the dura mater laid back over the cortex, and the wound closed after replacing



FIGS. 149, 150.—Antero-posterior and right lateral X-ray appearances of patient shown in *Fig. 148*. An opacity can be seen in the right frontal region.

the bone-flap. The epicranial aponeurosis was sutured separately with fine silk and the skin with interrupted silkworm-gut sutures. *Fig. 153* shows the X-ray appearances after operation.

FIG. 151.—Appearance of extradural plexiform hæmangioma at operation.

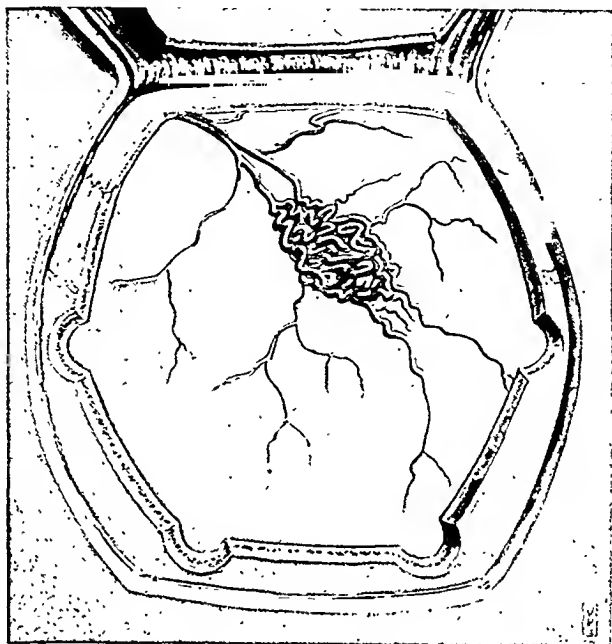
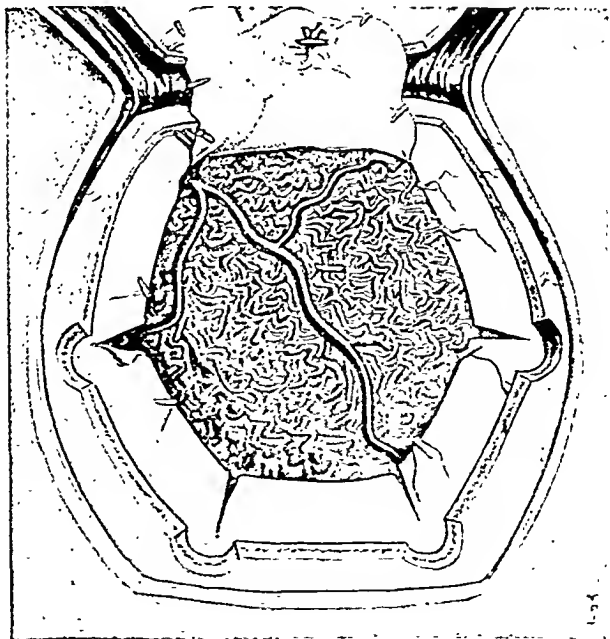


FIG. 152.—Appearance of intradural plexiform hæmangioma as revealed on opening the dura mater.



SECOND OPERATION.—Convalescence was uneventful except that two fits occurred, one three days and the other twenty-two days after the operation. Neither of these was severe and neither was followed by hemiplegia. It was thought advisable, however, to attempt further to interrupt the blood-flow through the lesion, and



FIG. 153.—X-ray appearances after operation. The position of the bone-flap, with the subtemporal decompression, is shown. The small dark shadows are made by silver clips occluding vessels.

accordingly after a few weeks in a convalescent home, the patient was re-admitted, and on April 1, 1932, the right internal carotid artery was occluded with two catgut ligatures applied just beyond its origin from the sinus caroticus.

PRESENT CONDITION.—The patient was discharged from hospital on April 13, and has been seen from time to time. There have been one or two fits of a mild character, but these have not been followed by hemiplegia and the boy has returned to school and is able to lead a normal life.

Commentary.—A case is here recorded of extensive extra- and intradural plexiform arteriovenous anastomoses connected with each other, and associated with a facial capillary hæmangioma (nævus) on the same side. The association between the facial and intracranial hæmangiomata is a well-recognized one, instances having been recorded in 1902 by Cassirer,¹ and since then by Harvey Cushing,² Hebold,³ E. Sachs,⁴ Dimitri,⁵ Marque,⁶ and others; but the very extensive character of the intracranial lesions in this case is unusual, as also is the fact that the intracranial lesion was apparently a serpentine arterial angioma, whereas the intracranial angiomata associated with facial nævi apparently are usually of the venous variety (Cushing and Bailey,⁷ 1928).

The clinical picture—namely, convulsions immediately followed by evanescent attacks of hemiplegia, and with little or no general progression of the

signs and symptoms—is characteristic of these intracranial blood-vessel lesions. Calcification, such as is shown in the X-ray plates of this case (see Figs. 149, 150), may or may not be present.

TREATMENT.

Two questions arise in connection with the treatment of these cases: (1) Is any treatment indicated? (2) If so, what should be its nature?

The answer to the first question would appear to depend upon certain subsidiary ones—namely: What is the outlook for the untreated patient, can this outlook be helpfully modified by treatment, and, finally, will such treatment be palliative as far as symptoms are concerned?

Life-history.—W. E. Dandy⁸ (1928) found that of 22 cases collected from reports, 9 had died of intracranial hæmorrhage, while 2 from his own group of 8 cases had had hæmorrhages from which they had recovered. Such patients, therefore, are faced with an ever-present danger of intracranial hæmorrhage, which if it takes place, is usually fatal. Ernest Sachs⁹ (1931) and others have recorded cases which have been successfully operated upon and relieved from symptoms. Sachs' case was a boy aged 10, with a facial port-wine stain, normal fundi, and attacks of convulsions. He was operated upon in February, 1915, an osteoplastic exploration being performed and occlusion of some of the vessels associated with the lesion, which from the description appears to have been somewhat similar to the one here reported. The patient was much improved, his fits diminishing and finally ceasing within a few months of leaving hospital. He then became and has since remained perfectly well. There would thus appear to be sound reason combined with precedent for operating upon such cases.*

The second question—What should be the nature of the treatment?—requires careful consideration. The temptation to do too much is great. Attempts to extirpate the vascular mass are to be deprecated. There may appear to be indications to apply many ligatures to the multitude of vessels comprising the lesion, but if such a procedure is followed, there is an immediate danger of hæmorrhage, which, if it occurs, may be difficult to control owing to the defective nature of the walls of the vessels; or if this danger is averted, there is the subsequent one of a persisting monoplegia or hemiplegia.

It is probably because of these considerations that Cushing and Bailey⁷ give as their summing-up to the question of treatment of the cerebral venous angiomata: "The lesions, in short, when accidentally exposed by the surgeon had better be left alone." In the case of certain of the arterial angiomata, however, they appear to favour radiotherapy, decompression, and carotid ligations. Sachs' case and the one here recorded would appear to justify a certain degree of guarded surgical interference, efforts being made to bring about partial vascular occlusion by means such as have here been described.

* E. Sachs has recently reported (*Southern Medical Journal*, 1932, Oct., xxv, 1013) a method of treating these cases by electrosurgery; the vessels are gently stroked by an electrode conveying a weak coagulating current. He points out that coagulating large vessels is tedious and must be done slowly, and that if the current used is too strong, the vessel being stroked is perforated and a large hæmorrhage occurs; but he has now been able to apply this method successfully in several cases.

THE NATURE OF THE LESION.

Such lesions as that presented by this case do not appear to represent true blastomatous tumours, but rather maldevelopments of the original vascular bed from which arteries, veins, and capillaries normally develop.

Dandy⁸ quotes from Virchow's *Krankhaften Geschwülste* to show that the great German pathologist was aware of the true nature of these lesions: "There remains that form of angioma in which the vascular enlargement is outspoken and in which the character of the tumour is more in the background . . . one cannot make a true line of demarcation between them and angiomata. Their difference lies principally in the fact that the process is diffuse and that dilatation of both arteries and veins is present." Regarding them as arterio-venous aneurysms, Dandy believes that the varying appearances which they present depend upon the arterial and venous channels concerned in their formation and the nature of the intervening vascular bed. Cushing and Bailey,⁷ however, while classifying the blood-vessel tumours of the brain in two groups—namely, the vascular malformations and the hæmangioblastomas—subdivide the former, which are most commonly found in the cerebral hemispheres, into teleangiectasis, angioma venosum, and angioma arteriale; and the latter, which are almost exclusively limited to the cerebellum, into cystic and solid varieties. The separation into two main groups is indisputable and very desirable, but the subdivision of the malformations would appear to be less so, since intermediate types occur between the three sub-varieties, and Dandy's explanation of the cause of these modifications appears to be a reasonable one and to reduce them to a common basis.

CONCLUSION.

It is possible at times to make a pre-operative diagnosis of intracranial blood-vessel tumour of the malformation variety. The symptoms produced by such a lesion may be alleviated, and the danger of fatal intracranial hæmorrhage averted, by osteoplastic exploratory craniotomy with occlusion of the principal source of blood-supply to the lesion.

REFERENCES.

- ¹ CASSIRER, R., "Angiom des Gehirns", *Neurol. Centralblatt.*, 1902, xxi, 32.
- ² CUSHING, H., "Cases of Spontaneous Intracranial Hemorrhages associated with Trigeminal Nævi", *Jour. Amer. Med. Assoc.*, 1906, xlvii, 178.
- ³ HEBOLD, O., "Hæmangiom der weichen Hirnhaut bei Nævus vasculosus des Gesichts", *Arch. f. Psychiat.*, 1913, li, 445.
- ⁴ SACHS, E., "Intracranial Telangiectasis: Symptomatology and Treatment, with Report of Two Cases", *Amer. Jour. Med. Sci.*, 1915, cl., 565.
- ⁵ DIMITRI, V., "Tumor cerebral congénito (Angioma cavernoso)", *Rev. Asoc. méd. argent.*, 1923, xxxvi, 63.
- ⁶ MARQUE, A. M., "Consideraciones sobre Angiomas en la Infancia", *Rev. Oto-Neuro-Oftalmol. y de Cir. neurol.*, 1927, i, 202.
- ⁷ CUSHING, H., and BAILEY, P., *Tumours arising from the Blood-vessels of the Brain. Angiomatous Malformations and Hæmangioblastomas*, 1928. London: Baillière, Tindall & Cox.
- ⁸ DANDY, W. E., "Arterio-venous Aneurysms of the Brain", *Arch. of Surg.*, 1928, Aug., 190.
- ⁹ SACHS, E., *The Diagnosis and Treatment of Brain Tumours*, 1931. London: Kimpton.

POUCHES OF THE PHARYNX AND ŒSOPHAGUS WITH SPECIAL REFERENCE TO THE EMBRYOLOGICAL AND MORPHOLOGICAL ASPECTS.

(The Arris and Gale Lecture delivered before the Royal College of Surgeons, Feb. 17, 1933.)

BY R. W. RAVEN, LONDON.

POUCHES of the pharynx and œsophagus have been subject to much speculation for nearly two hundred years. Contributions have been made by many orders of our profession—surgeons, physicians, morphologists, and embryologists. In spite of this there is probably no other subject in surgery in which so many inaccurate observations have been made or so many erroneous theories perpetrated. At the present day the literature is full of errors, and these are present even in the latest standard works of surgery. In this paper I wish to consider the subject especially from the embryological and morphological aspects. Certain facts already known will be emphasized, doubt will be cast on some views which are held, and several new varieties of pouches will be described.

CLASSIFICATION.

Pouches of the pharynx and œsophagus may be classified as follows :—

POUCHES OF THE PHARYNX.—

1. *Congenital* :

- a. Lateral pouch in connection with second embryonic endodermal pouch.
- b. Lateral pouch derived from third embryonic endodermal pouch.
- c. Lateral pouch derived from fourth embryonic endodermal pouch.

2. *Acquired* :

- a. Posterior pulsion.
- b. Anterior pulsion.

POUCHES OF THE ŒSOPHAGUS.—

1. *Congenital* :

- a. Associated with œsophago-tracheal fistula.
- b. Associated with imperfect separation of œsophagus and trachea.
- c. Associated with localized imperfect development of muscle coats.
- d. Associated with multiple pouches of the colon.
- e. Single pouch in the posterior wall.

2. *Acquired* :

- a. Tuberculous pouches.
- b. Secondary to œsophageal stricture.
- c. Secondary to œsophageal ulceration.

POUCHES OF THE PHARYNX.

CONGENITAL POUCHES.

Development of the Pharynx.—In all Craniota bilateral symmetrical pouches are formed from the anterior region of the foregut. At the same time corresponding invaginations form in the ectoderm. As a result of outward growth, the pouches press aside the lateral mesoderm of the head and come into apposition with the corresponding ectodermal invaginations

(*Fig. 154*). The temporary fusion of endoderm with ectoderm forms the epithelial closing membrane.

In animals with branchial respiration, clefts are formed by perforation of the epithelial closing membrane; the number of branchial clefts varies from five to nine in different forms. The closing membrane in the human embryo and in the majority of mammals remains imperforate, and open clefts do not normally occur.

As growth proceeds, dorsal and ventral angles are formed in each pouch with the exception of the first, and simultaneously the epithelial closing membrane elongates. The connection of each pouch with the pharynx becomes constricted owing to the disparity of growth between the lateral part and the medial part; this constriction forms the endodermal duetus pharyngo-brachialis.

The structure of the early pharynx is very transitory. The

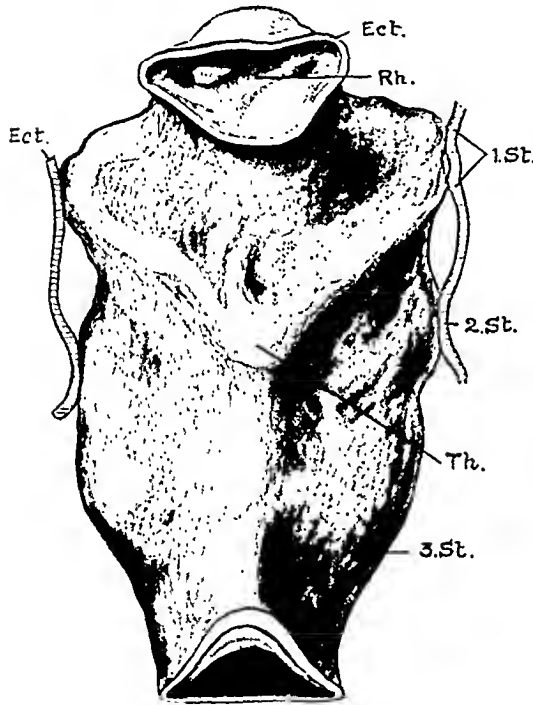


FIG. 154.—Pharynx of embryo about 3 mm. Ect., Ectoderm; Rh., Pharyngeal membrane; Th., Thyroid; St., 1st, 2nd, 3rd pharyngeal pouches. (After Keibel and Mall—"Human Embryology".)

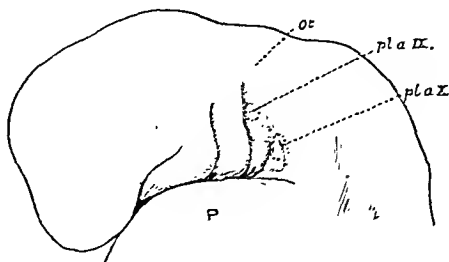
entire region is in a state of continuous growth. As a result of the proportionate size and growth of the outer pharyngeal arches, the pre cervical sinus appears on the surface at the early stage of 5 mm.

I wish to call attention to the broad strip of thickened ectoderm running from before backwards, along the upper ends of the arches and grooves (*Fig. 155*). This is the placodal area. In certain parts the cells of this area are continuous with the cellular masses of the nerve rudiments of the pharyngeal arches; two of these parts are indicated in *Fig. 155* by dotted lines and are the placodal areas of the ninth and tenth cranial nerves. The ninth nerve placode is situated at the upper end of the third arch, some distance down the side of the second groove.

The Ultimate Fate of the Ninth Nerve Placodal Area and its Connections.—The importance of the ninth nerve placodal area in the interpretation of certain vestigial structures in the neck has been overlooked.

The area of the preecervical sinus deepens markedly owing to the rapid growth of its boundaries. There is also a large increase in the paraxial mesoderm. Consequently the ninth placodal area comes to lie at the bottom

FIG. 155.—The early pharynx, showing: Ot., Otocyst; P, Pericardium; pl. a. ix., Placodal area of the glossopharyngeal nerve; pl. a. x., Placodal area of the vagus nerve. (After J. Ernest Frazer—*Jour. of Anat.*, lxi.)



of a short recess which opens into the upper part of the second groove. Later the ninth placodal area is completely covered and folded in, so that it lies at the enlarged end of a deep tunnel which opens on the surface. Very soon the placodal area is completely shut off from the surface, and there is formed a buried cyst in intimate relation with the endodermal wall of the pharynx and connected with the surface by ectodermal cells (*Fig. 156*). If growth is normal, the ectodermal cyst and duct disappear at 12 mm.

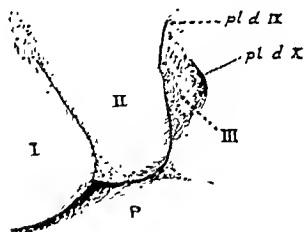


FIG. 156.—The early pharynx, showing: pl. d. ix., Placodal duct of the glossopharyngeal nerve; pl. d. x., Placodal duct of the vagus nerve; I, II, III, First, second, and third branchial arches; P, Pericardium. (After J. Ernest Frazer—*Jour. of Anat.*, lxi.)

Discussing the development of this region, Frazer states, "the placodal areas certainly stand forward as the structures that are mainly affected". It is conceivable that the ninth placodal cyst lying in close relation to the second pharyngeal pouch may persist along with the duct connecting it with the surface. This is the only connection between the skin and pharynx which has been proved to exist in embryonic life. The schematic representations of internal pharyngeal ducts opening into a large covered cervical sinus from which external ducts open on the surface is incorrect and does not rest on proved facts. In the schematic representations the only structures proved to exist are the endodermal pharyngeal pouches with their endodermal ducts, and the placodal cysts with their ectodermal ducts.

The relations of the endodermal pouches in the adult pharynx are fixed. The position of the second is at the tonsil, the third in the pyriform fossa, and the fourth at the lower end and lateral aspect of the pharynx.

Lateral Pouches.—It is unjustifiable in the present state of our knowledge to dogmatize in assessing the embryological significance of vestigial structures in the neck, and in presenting three lateral pouches of the pharynx I suggest the most likely modes of origin.

Case 1.—Boy, aged 16 years, under the care of Mr. Keynes, to whom I am indebted for permission to record the case. The patient had noticed a small hole in the right side of the neck for four years. On examination a small pin-point opening was seen at the junction of the upper two-thirds and lower third of the anterior border of the right sternomastoid. A cord one inch long was felt running upwards from the orifice. At operation a cyst was found in the right side of the neck with an external opening and an internal duct passing upwards and inwards under the posterior belly of the digastric muscle and between the internal and external carotid arteries. The duct opened into the pharynx immediately below and lateral to the right tonsillar fossa. Microscopic examination of the duct close to the pharynx showed a lining of squamous stratified epithelium.

In this specimen, therefore, there is a large pouch in the neck connected with the pharynx and skin by slender ducts. In embryonic life the only structures proved to connect endoderm and ectoderm in this region are the placodal cyst and duct in connection with the ninth cranial nerve. I regard this vestigial structure as a derivative of the ninth placodal cyst in relation with the second endodermal pouch.

Watson has described a similar pouch opening into the pharynx at the posterior faucial pillar whose walls were innervated by several branches from the ninth cranial nerve.

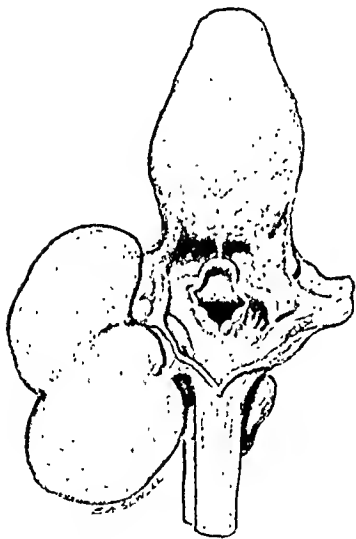


FIG. 157.—Case 2. Pouch on the left lateral aspect of the pharynx opening by a duct into the bottom of the left pyriform fossa. (Specimen in the Museum of Great Ormond Street Hospital for Children.)

Case 2.—Infant, aged 3 weeks, under the care of Mr. Eric Lloyd. I am indebted to the Medical Committee of Great Ormond Street Hospital for Children and to Mr. Lloyd for permission to study this specimen. The child was born with a swelling on the left side of the neck which caused choking attacks during feeding. At autopsy the condition shown in *Fig. 157* was found.

On examining the specimen I found a small round orifice at the bottom of the left pyriform fossa, and careful dissection revealed a small duct passing upwards and inwards from the cyst to the pharyngeal orifice. Microscopic examination

of the cyst wall showed a lining of granulation tissue, fibrous tissue with numerous inflammatory cells, and an external muscle coat.

I regard this as a derivative of the third endodermal pharyngeal pouch and duct lying in relation with the left pyriform fossa.

Case 3.—The specimen is from the Shattock Museum of St. Thomas's Hospital, studied through the kindness of Professor Dudgeon, and is shown in *Fig. 158*. Along the left side of the pharynx and œsophagus is a thin membranous pouch 3 in. long, which communicates with the lowest part of the pharynx by a circular aperture situated in the fibres of the inferior constrictor of the pharynx, immediately behind the posterior border of the thyroid cartilage. The neck of the pouch passes behind the termination of the common carotid artery.

From the anatomical relations of this pouch, it appears to be derived from the fourth embryonic endodermal pouch. The ultimate size of the pouch is due to repeated distension with food.

Importance of the Hypoglossal Nerve in relation to Embryonic Cervical Vestigial Structures.—Frazer has drawn attention to the importance of the course of the hypoglossal nerve in relation to these structures. The hypoglossal nerve in the beginning of its course lies behind the region of the arches

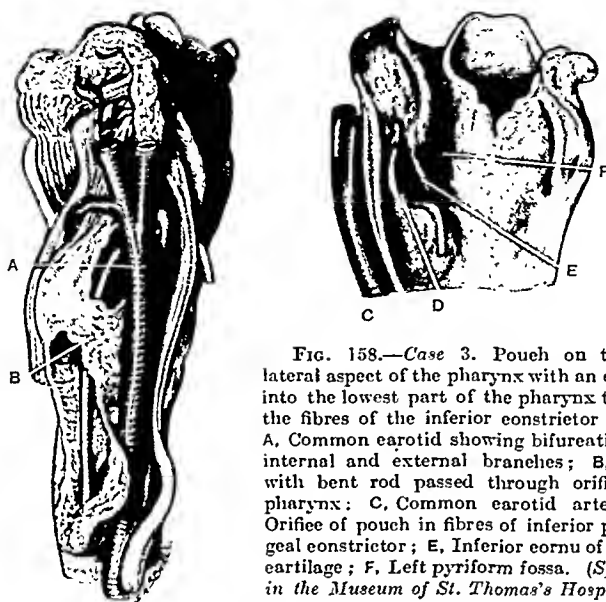


FIG. 158.—Case 3. Pouch on the left lateral aspect of the pharynx with an opening into the lowest part of the pharynx through the fibres of the inferior constrictor muscle. A, Common carotid showing bifurcation into internal and external branches; B, Pouch with bent rod passed through orifice into pharynx; C, Common carotid artery; D, Orifice of pouch in fibres of inferior pharyngeal constrictor; E, Inferior cornu of thyroid cartilage; F, Left pyriform fossa. (*Specimen in the Museum of St. Thomas's Hospital.*)

and grooves. In passing to its ultimate distribution the nerve is deep to structures connected with the ectoderm, and superficial to structures connected with the pharynx. Consequently any connection persisting between the surface and pharyngeal wall, or its derivatives, would be caught up over the hypoglossal nerve. On this account it is not surprising that few cervical vestiges have a deep connection, as this is usually severed by tension caused by the hypoglossal nerve.

ACQUIRED POUCHES.

Pharyngeal Diverticulum—Posterior, Acquired.—The first description of this condition is contained in a letter written in 1764 by Ludlow, of Bristol, to William Hunter. His method of examination was thorough, and included the introduction of a probang, whalebones, flexible catheters, and finally the patient was told to swallow a great quantity of quicksilver, and, as it did not enter the stomach, the surgeon was at a loss to localize it. However, the quicksilver was recovered at post-mortem from a large pharyngeal pouch.

Sir Charles Bell in 1816 first shed light on the etiology, by calling attention to spasmodic contraction at the œsophageal entrance followed by repeated

ineffectual attempts to swallow. Zenker and Von Ziemssen in their classical study of 1877 contributed to the morbid anatomy, symptomatology, and diagnosis of the condition.

ETIOLOGY.—Many theories have been advanced in regard to the pathogenesis of the condition. There is no evidence that the structure is atavistic in origin. Some authorities have likened it to the pouch which occurs in the domestic pig, but the morphology of this structure is entirely different. Various erroneous congenital theories have been advanced. It may be stated at once that the pouch is a prolapse of the mucous membrane of the pharynx in an area bounded by two different sets of musculature, innervated by different nerves. A consideration of the structure of this area surprises one that the condition is not more frequent.

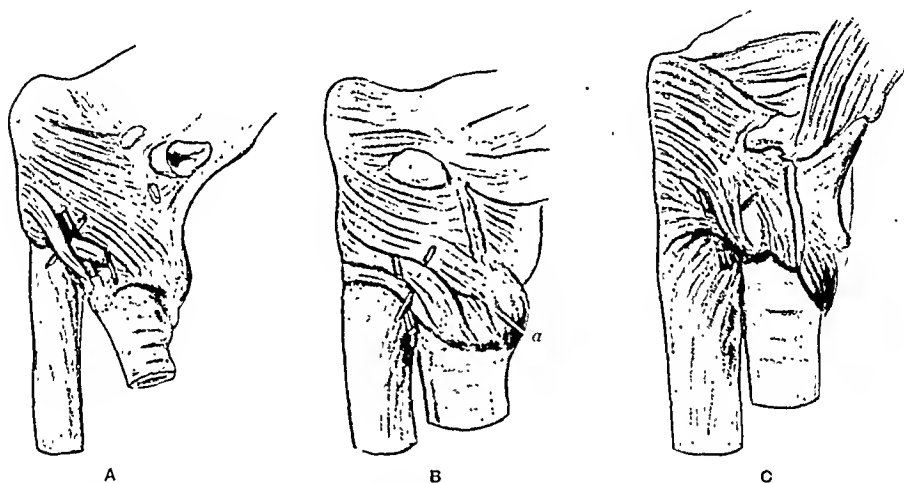


FIG. 159.—The arrangement of the fibres of the cricopharyngeus muscle in monkeys. A, The orang: the muscle is composed of superficial constrictor fibres; there is a special sphincteric muscle arising from the thyroid cartilage (red rod). B, The chimpanzee: the white rod (a) is under the superficial constrictor fibres; the red rod is under the deep sphincteric fibres. C, The silver gibbon: arranged as under B.

The Œsophageal Orifice in Different Species.—In the dogfish the œsophagus is not more than 3 in. in length and entirely composed of a muscular sphincter of strong circular fibres. In the crocodile the cornua of the hyoid bone guard the œsophageal entrance and form a well-marked sphincter. This mechanism is complicated still further in the tortoise. In mammalia the hyoid bone no longer controls the œsophageal entrance; the cricoid cartilage now forms the basis of the sphincteric mechanism. Negus has shown that such a mechanism is necessary in all lung-breathing forms to prevent air suction into the œsophagus during respiration. The ornithorhynchus has a well-marked band of muscle encircling the œsophageal entrance, forming a sphincter with a weak attachment to the fused cricothyroid ring. The arrangement of the muscles in monkeys is shown in Fig. 159.

In the human fœtus the cricopharyngeus muscle is divided into an upper superficial constrictor part, incorporated in the pharyngeal constrictor musculature, and a lower deeper sphincteric part which blends posteriorly

with the muscle of the œsophagus. In a four-months' fœtus (*Fig. 160A*) this division is evident, and the constrictor portion passes obliquely upwards whilst the sphincteric part passes transversely round the œsophagus. In a five-months' fœtus (*Fig. 160B*) the direction of the sphincteric portion is obliquely downwards. Consequently a weak area is produced in the posterior wall of the pharynx between the superficial and deep portions of the ericopharyngeus muscle. It is through this weak area that the pharyngeal mucosa prolapses in the adult (*Fig. 161*). A fully formed pouch is shown in *Fig. 162*.

I have satisfied myself in examining a large number of these pouches that the mucous membrane of the pharynx only herniates through this area.

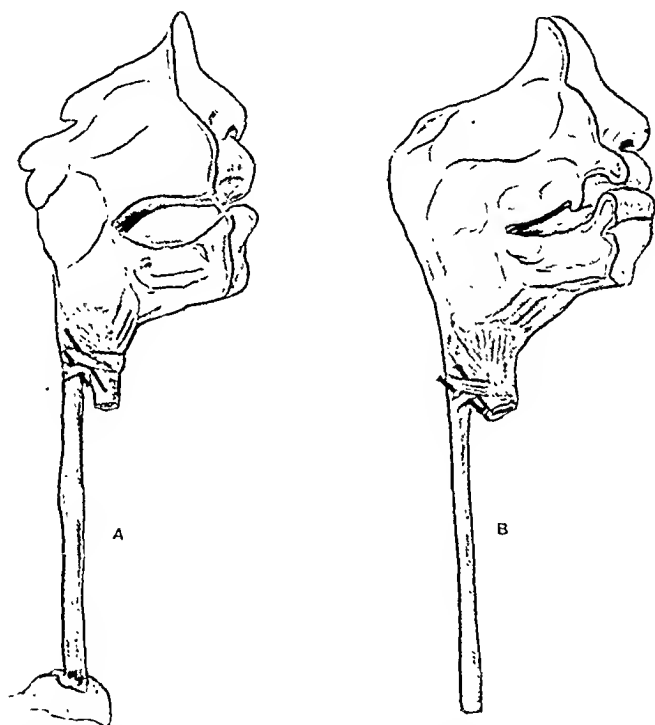


FIG. 160.—The arrangement of the ericopharyngeus muscle in the fœtus. A, Four months; B, Five months. The black rod is under the superficial constrictor fibres; the red rod is under the superficial constrictor and deep sphincteric fibres.

The cricopharyngeus muscle therefore consists morphologically and functionally of two distinct sets of musculature with a congenital weakness between them. If either of the two following conditions is present, prolapse of the mucous membrane will occur.

1. Loss of elasticity or degeneration of the muscles composing the lower pharyngeal constrictors. At best, this musculature is poor material, and even under normal conditions this region is one of stress and strain.
2. Persistence of contraction of the sphincteric portion of the ericopharyngeus muscle during deglutition is probably the more important factor.

We know this occurs from the histories of patients with pouches. Consequently the intrapharyngeal pressure increases and the weakest part of the wall gives way.

During the second stage of normal deglutition the pharyngeal constrictors contract, and simultaneously the sphincteric portion of the cricopharyngeus muscle relaxes. This is a complex and involuntary reflex action. Why the sphincter fails to relax in certain individuals we do not know. It is feasible to suggest that it is due to a breakdown in the neuromuscular mechanism. We are familiar with such breakdowns in certain other regions of the alimentary canal, but as yet we do not know the site of the primary lesion, whether it is central, ganglionic, peripheral, or due to some internal secretion.

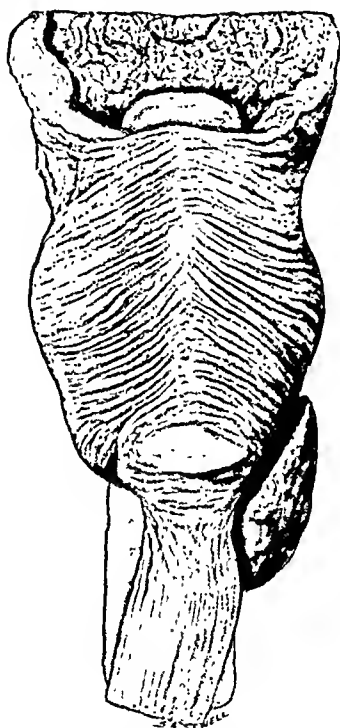


FIG. 161.—Early prolapse of pharyngeal mucosa between the two parts of the cricopharyngeus muscle. (*Specimen in the Museum of the Royal College of Surgeons.*)

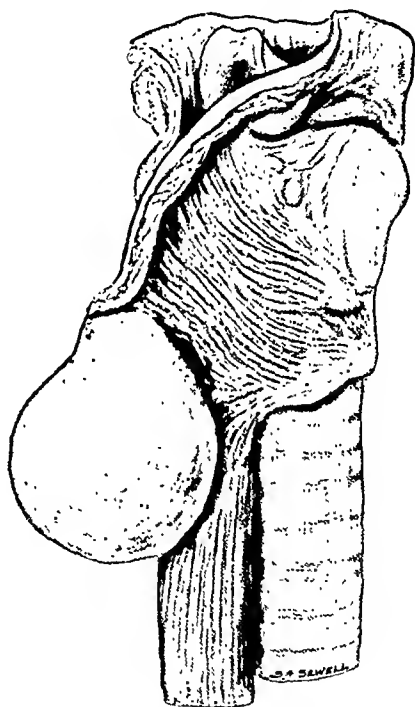


FIG. 162.—Pharyngeal pouch. The two parts of the cricopharyngeus muscle are seen. (*Specimen kindly lent by Mr. Carthorne.*)

The nerve-supply around the œsophageal entrance is complex. The pharyngeal constrictors, including the constrictor portion of the cricopharyngeus, are supplied by the intricate pharyngeal plexus. The upper end of the œsophagus, including the sphincteric part of the cricopharyngeus, is supplied by numerous motor nerves from the recurrent laryngeal nerves. Contributions to the innervation of this region are also made by the sympathetic system, but the rôle of these nerves is obscure. The association of goitre with this type of pouch has been noted by German writers and is of interest in connection with derangement of the neuromuscular mechanism. It has

been suggested that the enlarged thyroid gland raises the intrapharyngeal pressure by pressing on the œsophagus and thus herniation of the pharyngeal mucosa occurs. It is more likely, however, that the motor nerves to the œsophageal orifice are mechanically stimulated by the enlarged thyroid gland, with consequent spasm of the sphincter. I suggest the possibility of œsophageal spasm in these cases being due to increased excitability of the vagosympathetic nerves to the sphincter, caused by deranged thyroid metabolism.

Pharyngeal Pouch—Anterior, Acquired.—This type of pouch is very rare. Hurst and Briggs have recorded an example in a female aged 57 years which was discovered by radiological methods immediately in front of the entrance to the œsophagus and behind the larynx.

POUCHES OF THE ŒSOPHAGUS.

CONGENITAL POUCHES.

Pouch Associated with an Œsophago-tracheal Fistula.—The marked constancy of the morphology of this condition has been recognized by numerous observers and is a sign of an early fundamental change in the embryo, as changes occurring late in embryonic life give rise to anomalies which do not conform to a definite pattern. There may be a genetic basis for this malformation, as Mackenzie found it present in all the children of one father by three wives.

The pathological picture is very constant (*Fig. 163*). The œsophagus commences in the usual way and ends blindly, forming a uniformly dilated pouch with thin walls. The large size of the pouch may be due to distension with amniotic fluid or to an abnormal growth stimulus. In most cases the lower portion of the œsophagus opens into the trachea at the bifurcation or a short distance above.

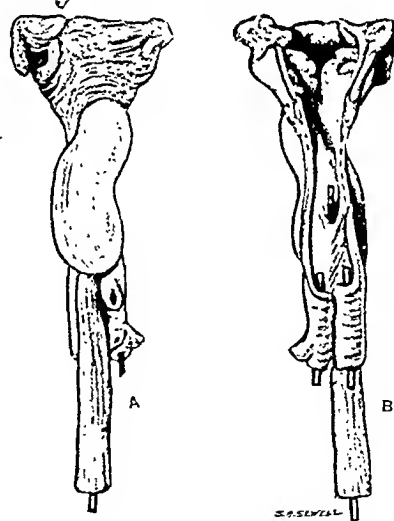


FIG. 163.—Œsophago-tracheal fistula associated with a pouch of the upper end of the œsophagus. A, Posterior aspect; B, Anterior aspect.

Numerous theories have been put forward to account for this anomaly. Malformation of the œsophago-tracheal septum has been widely accepted, but as we do not know precisely how the œsophagus and trachea separate, the theory does not satisfy. Others have advanced theories of intra-uterine inflammation and intra-uterine trauma.

It is not surprising that congenital anomalies should occur sometimes, when we consider the intricacies of the developmental process, each species negotiating the various stages at specific rates, which vary within certain normal limits. If these normal limits are transgressed, the product of development is distorted. Stockard has performed experiments in which the rate

of development was modified at will and various congenital malformations were produced. He showed that temporary arrest of growth at certain critical moments in development is followed by disastrous results. Stockard states that practically any deformity recorded in the literature, other than those resulting from germinal variations or mutations, may be induced by lowering the temperature, which modifies the rate of growth. The malformation under review, therefore, may be due to changes in the rate of growth in the cells which normally separate the trachea from the œsophagus. The exact nature of the forces causing these changes is little understood at present. Changes in temperature and environment may play a prominent part.

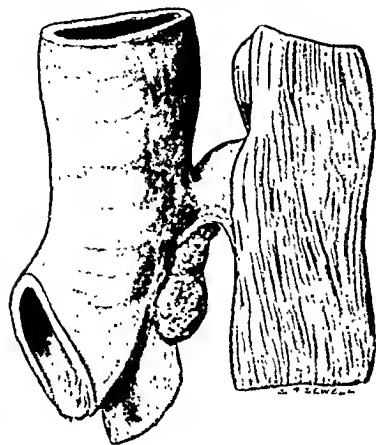


FIG. 164.—Pouch in anterior wall of œsophagus above the bifurcation of the trachea. The wall of the pouch is intimately incorporated in the wall of the trachea. The lymph-gland is merely attached to the lower border of the pouch. (Specimen in the Museum of the Royal College of Surgeons.)

Œsophageal Pouch due to Imperfect Separation of the Trachea and Œsophagus.—

Pouches of this nature occur in the anterior wall of the œsophagus a short distance above the bifurcation of the trachea or in the angle of bifurcation. Careful comparison with pouches due to adherent tuberculous lymph-glands show marked differences.

Whereas pouches due to inflamed lymph-glands are small and taper towards the apex, to which a mass of lymph-glands are attached, with the long axis placed obliquely upward or downward according to the direction of the glandular pull, the pouch under consideration

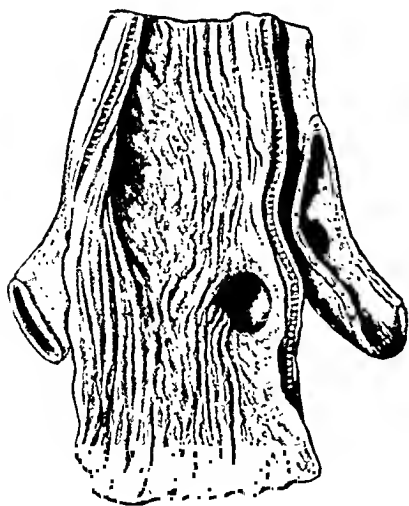


FIG. 165.—Pouch in anterior wall of œsophagus with the upper wall incorporated in the wall of the trachea. There is a depression in the right bronchus corresponding with the fundus of the pouch. (Specimen in the Museum of St. Bartholomew's Hospital.)

is round, with a broad fundus and circular orifice, with the long axis at right angles to the œsophagus, or obliquely upward towards the trachea or bronchus (*Figs. 164, 165*). Moreover, a part of the wall of the pouch adjoining the trachea is firmly incorporated in the tracheal wall. The muscle bundles are clearly demarcated on each side of the pouch.

If development is pursuing a normal course, the œsophagus and trachea separate at 5 mm. and the musculature of the tubes is formed at 10 mm. Hence the primary defect in the wall must be epithelial. In this connection Ribbert has demonstrated primitive ciliated cylindrical epithelium in the walls of these pouches.

In a series of transverse sections of a 17-mm. human embryo kindly lent to me by Professor J. E. Frazer, there is imperfect separation of the trachea and œsophagus with epithelial connection between the two tubes in three sections (*Fig. 166*). Such a defect may manifest itself in adult life as an œsophago-tracheal fistula, or partial closure may occur and the epithelium of the œsophagus become incorporated in the wall of the trachea at the site of the original fistula. Further development of the pouch is due to tension at the site of union due to elongation of the œsophagus, a certain degree of rotation which the œsophagus undergoes when the stomach rotates, and longitudinal contraction and relaxation of the whole œsophagus.

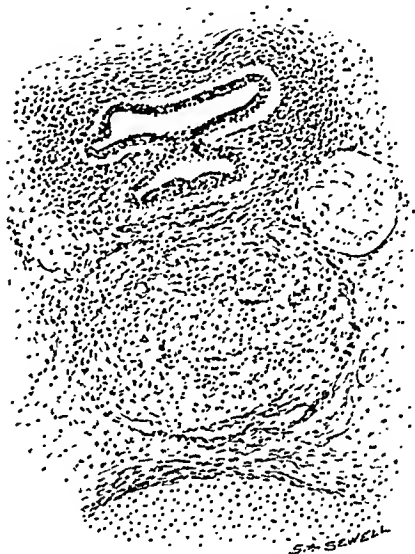


FIG. 166.—Transverse section of 17-mm. human embryo showing epithelial connection between the trachea and œsophagus. The position of the œsophagus is shown by the solid cord of cells. (*Serial sections of an embryo kindly lent by Professor J. E. Frazer.*)

Œsophageal Pouch Associated with Defect in the Muscular Coats.—

According to Keith the œsophagus is of double origin: the upper or para-tracheal part is derived with the trachea from the retropharyngeal segment of the foregut, and the lower or retropharyngeal part arises from the pre-gastric segment of the foregut.

I have noticed the marked difference in the upper and lower halves of the œsophagus in animals, the former being largely a membranous tube and the latter a strong muscular tube. In a four months' foetus the muscle coat of the lower half is well developed, whilst the upper half remains membranous. In the main, the muscle is unstriated in the lower half and striated in the upper half. Oppel regards the striated muscle as derived from the branchial muscle which arises from the lower head myotomes.

Fig. 167 shows a pouch of this nature occurring immediately below the bifurcation of the trachea, composed entirely of mucous membrane which does not bulge. The muscular defect is round, with clean-cut edges encircling



FIG. 167.—Pouch in anterior wall of the oesophagus below the bifurcation of the trachea.



FIG. 168.—Two small pouches below the bifurcation of the trachea. (*Specimen in the Museum of St. Bartholomew's Hospital.*)

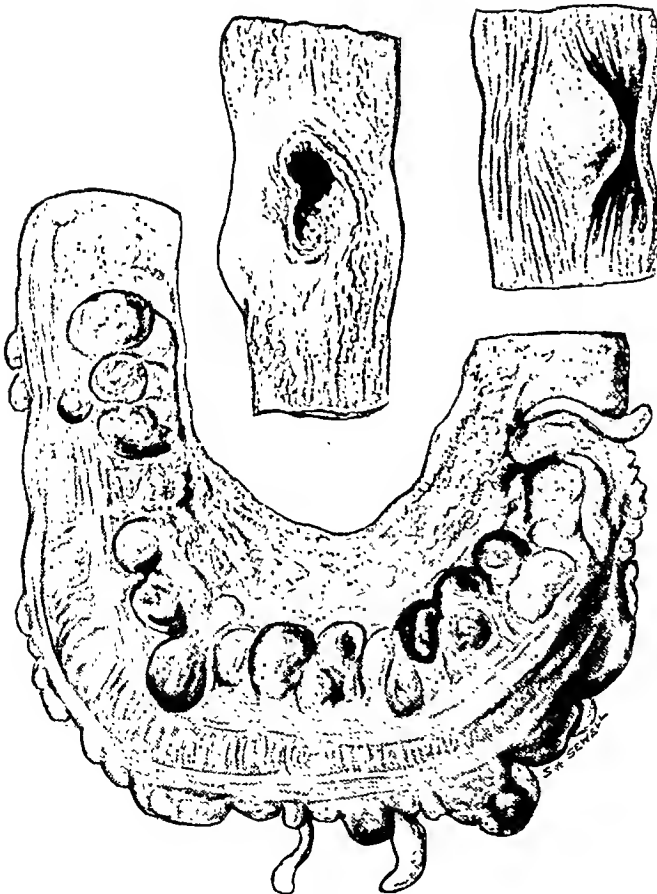


FIG. 169.—Oesophageal pouch associated with multiple diverticula of the colon. (*Specimen in the Museum of Charing Cross Hospital.*)

the pouch. The two lymph-glands have no connection with the pouch. In *Fig. 168* there are two small pouches immediately below the bifurcation of the trachea; the other coats of the Œsophagus are absent.

It is obvious that this type of pouch is not due to traction or pulsion, and I consider it to be due to a primary muscular defect in the region of union between muscle derived from the lower head myotomes and unstriated muscle developing *in situ*. Such muscular defects have been described in embryos of various ages. Hæppich found that the circular muscle in embryos of three and four months was completely interrupted in small areas. Shridde found a larger muscular defect in the Œsophageal muscle in contact with the trachea.

Œsophageal Pouch Associated with Multiple Diverticula of the Colon (*Fig. 169*).—The association of Œsophageal diverticula with those in other regions of the alimentary canal has been noted by Morrison and Smelt. The view is gaining ground that acquired diverticula of the whole intestinal tract from the Œsophagus to the anus owe their origin to similar tendencies and are local variations of the same morbid process. Barsony and Polgar have described multiple Œsophageal diverticula which they consider to be due to disturbed innervation.

Keith regards colonic diverticula as the manifestation of irregular contractions of the circular muscle of the colon due to disturbed innervation. This may be true for the colonic and certain multiple Œsophageal diverticula, but it is difficult to apply this theory to the example under review. Lewis and Thyng have demonstrated knob-like intestinal diverticula occurring regularly in embryos of man, rabbit, and pig, which usually degenerate but sometimes persist, in various regions. In connection with this specimen it is reasonable to suppose the presence of such congenital defects with the manifestation of well-marked pouches in later life.

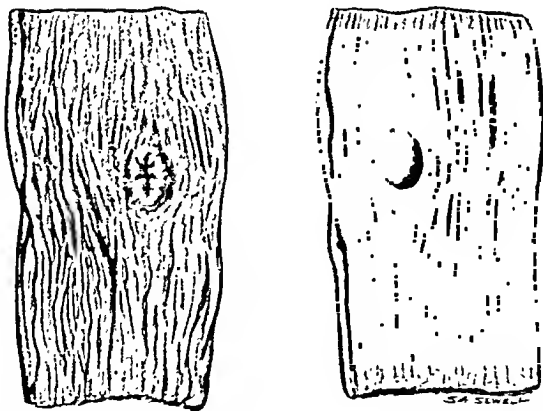


FIG. 170.—Pouch in the posterior wall of the Œsophagus. (*Specimen in the Museum of St. Bartholomew's Hospital.*)

Pouch in the Posterior Wall of the Œsophagus.—Pouches in the posterior wall of the Œsophagus are rare, and the appearances differ from all we have already discussed (*Fig. 170*).

The character of the pouch suggests that it may have originated in a cyst which has acquired a lumen into the œsophagus. Cysts of the œsophagus probably arise early in embryonic life from two sources. In human embryos

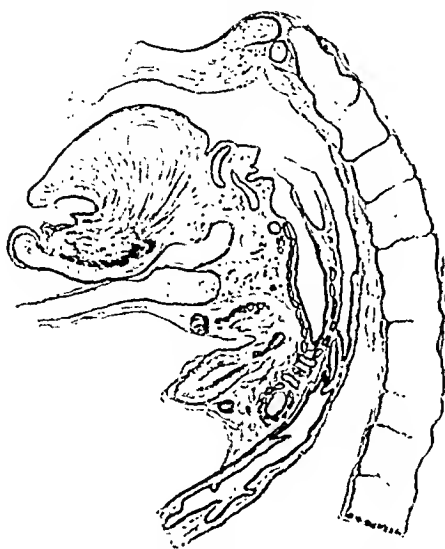


FIG. 171.—Diagram reconstructed from serial sagittal sections of a 10-weeks embryo, showing the developing œsophagus.

about 20 mm. vacuoles of various sizes occur in the œsophageal epithelium. In some places they open directly into the lumen, in others they are separated from it by a partition of epithelium. The vacuoles are more numerous in the lower half of the tube and in the anterior and posterior walls. Vaeuolation increases the size of the lumen of the œsophagus. These epithelial cavities may persist, forming either cysts or diverticula.

In later embryonic life the epithelium of the œsophagus is markedly irregular (*Fig. 171*). In circumscribed areas epithelial proliferation is taking place and epithelial bridges across the lumen occur. In other areas there is an active moving apart of the cells, resulting in well-marked depressions. These depressed areas may be isolated from the main lumen and covered in

by proliferating epithelium. Such cavities may remain closed, forming cysts, or may secondarily acquire an opening into the œsophagus, forming a diverticulum.

ACQUIRED POUCHES.

œsophageal Pouch due to Adhesion with Tuberculous Lymph-glands.—Kragh has made important contributions to the pathology of this type of pouch and prefers the term 'tuberculous diverticulum'. The pouch is conical in shape with an oval orifice with the long axis obliquely upward or downward. The muscle coat of the œsophagus seldom stops at the margin of the diverticulum. The apex of the pouch is firmly attached to diseased lymph-glands. The commonest site for their occurrence is in the anterior wall of the œsophagus in the area following the bifurcation of the trachea, where the œsophagus is in relation with the bronchial lymph-glands (*Fig. 172*).

Adhesions between lymph-glands and the œsophageal wall are not frequent; Kragh found adhesions in 14 out of 556 cases examined.

This type of pouch is uncommon in children. Dr. Allan Brown kindly searched the records of the large Children's Hospital in Toronto for me and could find no example. Mediastinal tuberculous lymphadenitis is more common in Toronto than in this country.

Pouches Associated with Obstruction at the Lower End of the œsophagus.—Pouches associated with cardiospasm are found in two regions—namely,

FIG. 172.—Pouches in the œsophagus due to lymphadenitis. (Specimen in the Museum of the Royal College of Surgeons.)

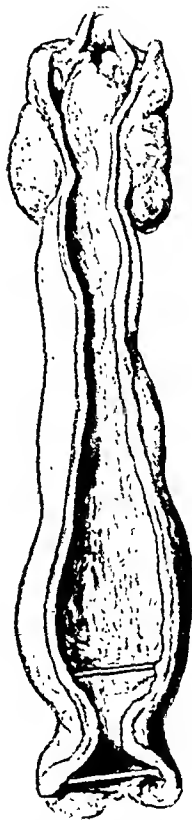
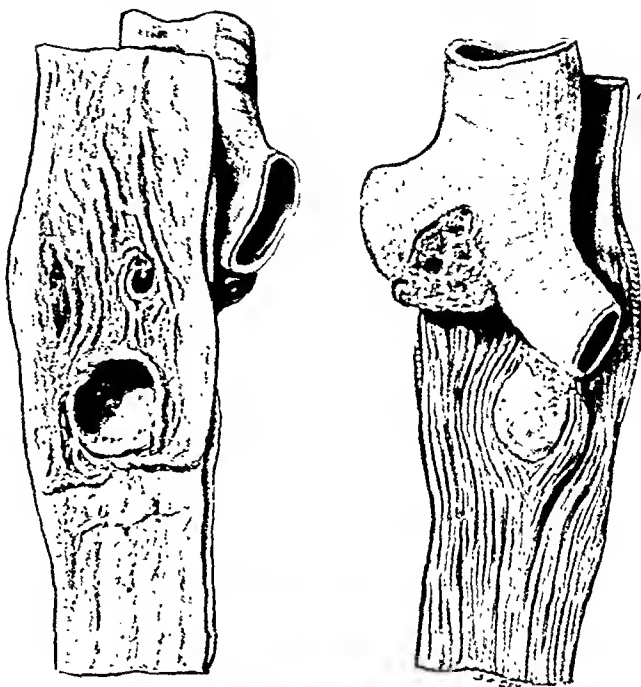


FIG. 173.—Fusiform pouch associated with stricture of the terminal œsophagus in a child. (Specimen in the Museum of Great Ormond Street Hospital for Children.)



FIG. 174.—Large pouch in the lateral wall of the œsophagus. The lumen of the lower end of the œsophagus is much diminished. (Specimen in the Museum of the Royal College of Surgeons.)

between the fibres of the cricopharyngeus muscle and in the lower third of the œsophagus. Twelve cases of pharyngeal pouch secondary to cardiospasm have been reported in the literature. Dessieker believes that a history of cardiospasm may be elicited in the majority of cases of pouches in the lower third of the œsophagus.

Pouches due to an organic stricture at the lower end of the œsophagus are fusiform or saccular. In the former the œsophagus is dilated throughout a localized area proximal to the stricture (*Fig. 173*). Abel has recorded an example of a saccular pouch situated above a stricture.

The effects of a stricture at the lower end of the œsophagus may be either uniform dilatation of the whole œsophagus, or partial dilatation with the formation of a fusiform pouch, or the muscle bundles may diverge in a localized area with consequent herniation of the mucous membrane (*Fig. 174*).

Œsophageal Pouch of Inflammatory Origin.—The relation of pouches and ulcers has excited interest. Perey and Shaw have drawn attention to the connection in the duodenum. *Fig. 175* shows a diverticulum in the right postero-lateral segment of the œsophagus $\frac{1}{4}$ in. above the cardiac orifice, with a wide oval orifice whose long axis is in the line of the œsophagus. The overlying muscle is absent and there is evidence of œsophagitis. It appears that this pouch is of inflammatory origin and very probably originated in an ulcer.

FIG. 175.—Pouch in the right lateral segment of the œsophagus. (Specimen in the Museum of St. Bartholomew's Hospital.)

NATURAL POUCHES IN ANIMALS.

The Sloth Bear.—There are two nasopharyngeal pouches, which Mayer described in 1830 (*Fig. 176*). According to Killian, they are not homologous with any structure in the pharynx of man.

Domestic Pig.—The nasopharyngeal pouch of this animal was described by Mayer in 1840. It is a large sac passing downward and backward from the lower end of the nasopharynx (*Fig. 177*). The orifice is guarded by a prominence formed by the lower part of the plica pharyngopalatini. The apex commonly projects backwards between the two divisions of the

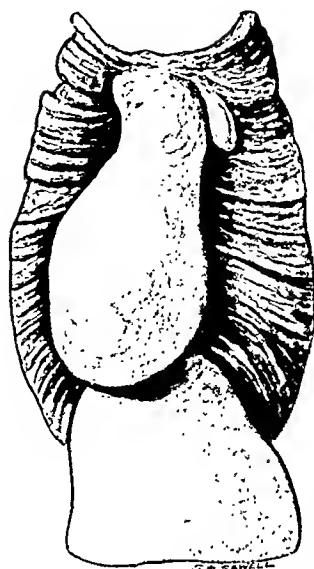


FIG. 176.

FIG. 176.—Nasopharyngeal pouch of the sloth bear. (*Specimen in the Museum of the Royal College of Surgeons.*)

FIG. 177.—Nasopharyngeal pouch of the domestic pig.

FIG. 178.—Anterior pharyngeal pouch of the great anteater.

FIG. 179.—Upper and lower œsophageal pouches of the fruit bat.



FIG. 177.



← Upper œsophageal pouch



FIG. 178.

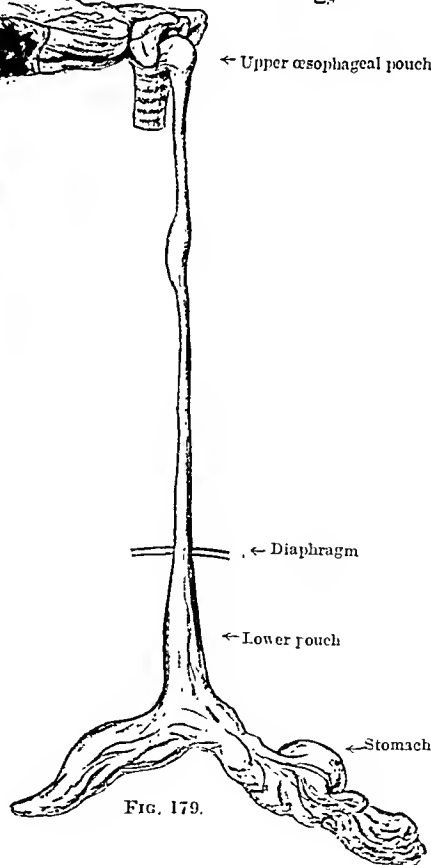


FIG. 179.

thyropharyngeus muscule. The upper part of this muscule acts as a loose sling, and the lower part is firmly blended with the inferior border of the pouch. An oblique muscule on each side passes deep to the thyropharyngeus to an attachment at the apex of the pouch. There is thus concerted muscular action at the apex to prevent excessive increase in size. The lower part of the thyropharyngeus muscle is divided into superficial and deep portions continuous above, and there is no space through which a pharyngeal pouch could occur as in man. It is clear that the morphology of this pouch is entirely different from the posterior pulsion pouch in man.

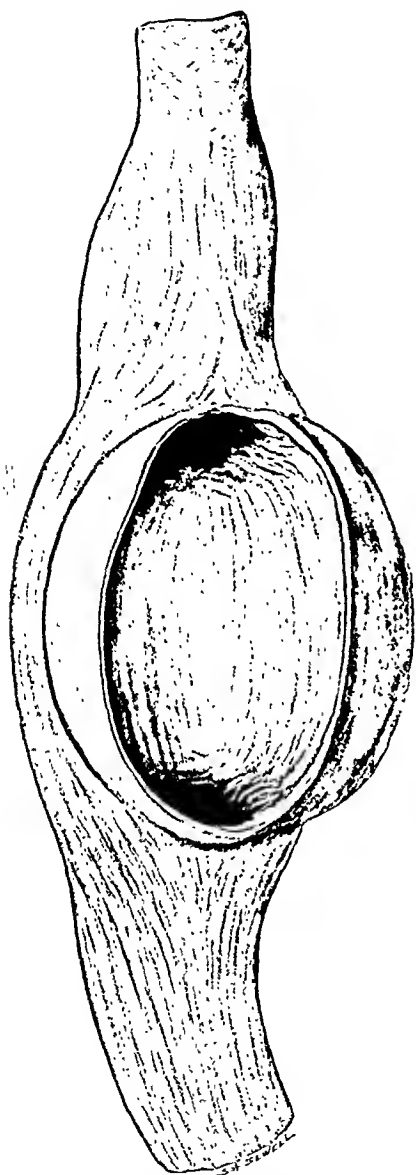


FIG. 180.—Large œsophageal pouch in a horse. (Specimen in the Museum of the Royal College of Surgeons.)

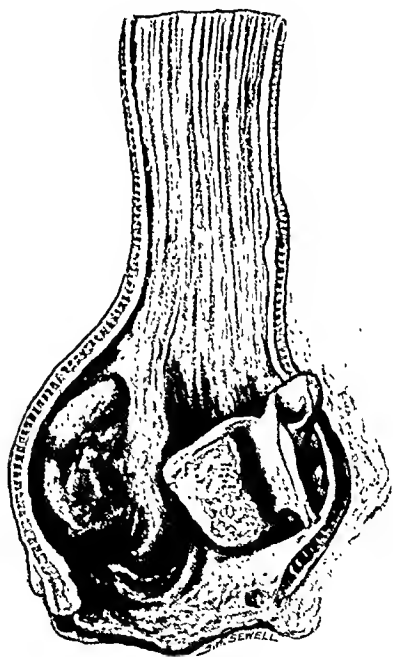


FIG. 181.—Pouch at lower end of œsophagus in a dog, containing a foreign body. (Specimen in the Museum of the Royal Veterinary College.)

Pharyngeal Pouches.—These are uncommon in animals. *Fig. 178* shows an anterior pharyngeal pouch which I removed from a great anteater. The membranous sac projects upwards and forwards above the hyoid bone. The orifice lies in the pharynx above the base of the epiglottis. It is difficult to

assign a function to this pouch. It is certainly not for the storage of food, and I suggest it secretes mucus for the lubrication of the food passages.

Œsophageal Pouches.—In the fruit bat I found a pouch at the upper end of the Œsophagus which has escaped the notice of earlier observers (*Fig. 179*). The pouch was present in three animals I examined, and consisted of a rounded sac situated between a tight circular sphincter mechanism above and weaker constrictor muscle fibres below. This animal spends many hours hanging head downwards from trees, and since there is no sphincter at the lower end of the Œsophagus, fluid runs up the tube, but is prevented from entering the pharynx by the strong sphincter at the junction of pharynx with Œsophagus. Hence the pouch is a pressure pouch, and the manner of production, lying as it does between a tight sphincter and a weak constrictor, is remarkably similar to the mechanism of production of the pressure pouch of man. Immediately below the diaphragm there is a fusiform pouch which is morphologically part of the stomach.

Pathological Pouches in Animals.—Pathological pouches in the pharynx of animals are rare, as they are not systematically looked for. In monkeys there is no reason why they should not occur, except in the orang, where the lower end of the pharynx is strengthened by a specialized muscular band. Negus has recorded a pharyngeal pouch occurring in a mangabey ape.

Pathological pouches commonly occur in the Œsophagus of animals. The horse is specially prone to pouch formation owing to rupture of the muscle coats (*Fig. 180*). Pouches occur frequently in the dog (*Fig. 181*).

I wish to thank the Council for the privilege of delivering this Arris and Gale Lecture. My best thanks are due to Mr. Burne for much kindness and help and to Mr. Wilson. I gratefully acknowledge the help of Professor G. E. Gask, Colonel Argyle, Sir Thomas Dunhill, Professor J. Ernest Frazer, Mr. Carwardine, Mr. Capps, Dr. Finzi, Dr. Sparks, and Dr. Simon, and the Curators of many hospital museums.

BIBLIOGRAPHY.

- MOYNIHAN, LORD, "Diverticula of the Alimentary Canal", *Lancet*, 1927, i, 1061.
 MAES, U., "Pharyngo-Œsophageal Diverticula: General Considerations", *New Orleans Med. and Surg. Jour.*, 1929, lxxxii, 126.
 JUND, E. S., and MOERSCH, H. J., "Pharyngo-Œsophageal Diverticulum", *Surg. Clin. N. Amer.*, 1929, ix, 793.
 PUMPELLY, W. C., "Pharyngo-Œsophageal Diverticulum: Case", *Southern Med. Jour.*, 1928, xxi, 1021.
 EVERINGHAM, S., "Pharyngo-Œsophageal Diverticula", *Surg. Clin. N. Amer.*, 1927, vii, 1343.
 JACOB, M., "Pharyngo-Œsophageal Diverticula and their Surgical Treatment", *Lyon méd.*, 1927, cxxxix, 199.
 HILL, W., "Pharyngeal and Œsophageal Diverticula", *Brit. Med. Jour.*, 1926, ii, 1163.
 ANGLIO, B., "Contributo allo Studio dei Diverticoli faringo-esofagei", *Arch. ital. di Chir.*, 1924, ix, 469.
 VERNIEUWE, "Diverticules multiples de l'Hyopharynx", *Bull. d'Oto-rhino-laryngol.*, 1924, n.s. xxii, 20.
 VEN DEN WILDENBERG, L., "Diverticules du Pharynx et de l'Œsophage", *Ann. des Mal. de l'Oreille*, 1922, xli, 783.
 FINZI, N. S., "Pharyngeal Pouches", *Arch. of Radiol. and Electrotherap.*, 1917, xviii, 342.
 VOL. XXI—NO. 82.

- WILKIE, D. P. D., and HARTLEY, J. N. J., "Pharyngeal Diverticulum and its Surgical Treatment", *Brit. Jour. Surg.*, 1922, x, 81.
- MIXTER, S. J., "Congenital Œsophageal Pouch", *Trans. Amer. Surg. Assoc.*, 1895, xiii, 357.
- KEIBEL and MALL, *Manual of Human Embryology*, 1912.
- RIEBOLD, G., "Œsophageal Diverticula", *Virchow's Arch.*, 1908, cxcii, 126.
- HALSTEAD, A. E., "Diverticula of the Œsophagus", *Ann. of Surg.*, 1904, xxxix, 171.
- LUDLOW, "A Case of Obstructed Deglutition from a Preternatural Dilation of, and Bag formed in, the Pharynx", *Medical Observations and Enquiries*, 1764, iii, 85.
- KILLIAN, G., "Ueber die Bursa und Tonsilla pharyngea", *Morph. Jahrb.*, 1888, xiv, 618.
- HURST, A. F., "Two Cases of Diverticula from the Lower End of the Œsophagus", *Guy's Hosp. Rep.*, 1925, lxxv, 361.
- HURST, A. F., and BRIGGS, P. J., "Anterior Pharyngo-œsophageal Pouch as a Cause of Dysphagia", *Ibid.*, 367.
- BUTLIN, SIR HENRY, "On the Removal of a 'Pressure Pouch' of the Œsophagus", *Med.-Chir. Trans.*, 1893, lxxvi, 269.
- GODLEE, R. J., and BUCKNALL, "A Pharyngeal Pouch of Large Size removed by Operation", *Lancet*, 1901, i, 1387.
- BELL, SIR CHARLES, "A Preternatural Bag formed by the Membrane of the Pharynx", *Surg. Obscr.*, 1817, i, 64.
- KRAGH, J., "Tuberculous Diverticula of the Œsophagus", *Acta Oto-Laryngol.*, 1922-3, iv, 49.
- KOSTANECKI, "Congenital Gill Fistulæ in Man", *Arch. f. Pathol. Anat.*, 1890, cxxi, 247.
- KREUTER, E., "Congenital Narrowings and Closures of the Intestinal Canal in the Light of Embryology", *Deut. Zeits. f. Chir.*, 1905, lxxix, 1.
- FLETCHER, H. MORLEY, "A Congenital Cyst of the Mediastinum", *Trans. Pathol. Soc. Lond.*, 1897, xlviii, 249.

THE SURGICAL TREATMENT OF BRONCHIECTASIS.*

By ROBERT M. JANES, TORONTO.

ATTEMPTS to relieve cases of bronchiectasis by surgical intervention have been, on the whole, discouraging. A fresh stimulus to the study of the disease was given by the introduction of lipiodol by Forrestier in 1922: before the employment of this diagnostic agent it was impossible, in the majority of cases, to be certain of the existence of bronchiectasis, and the accurate localization of the disease was out of the question. In a series of 72 cases Hartrung (quoted by Hedblom¹) was able by ordinary methods and röntgenograms to make a correct diagnosis as to the presence and site of the lesion in only 20; in 39 it was impossible to recognize either the lesion or its location. Under such circumstances great surgical progress was not to be expected.

Surgical methods of treatment may be divided into three main groups: (1) Collapse therapy as obtained by pneumothorax, section or exeresis of the phrenic nerve, or thoracoplasty; (2) Pneumotomy with drainage of the larger collections of pus; and (3) Excision or destruction of the involved lung tissue.

Collapse Therapy.—The pathological picture presented by the disease would seem to make it obvious that no form of collapse therapy could accomplish more than an alleviation of symptoms. The extent of the area in which secretions can collect may be decreased, but the underlying infection must still persist in the large majority of cases, and in very few is there a marked permanent reduction in the calibre of the dilated bronchi.

Oakley² recorded the results of *phrenic paralysis* in 17 cases of bronchiectasis. Complete relief of symptoms was reported in 4 cases (23·5 per cent), lasting improvement in 7 (41 per cent), and temporary improvement with relapse in 6 (35·5 per cent). Hedblom¹ reported 38 cases in his own experience, in which following phrenicotomy 9 were free of symptoms for from three months to three years, 20 had a decrease of 50 to 80 per cent in the amount of sputum, 7 showed some improvement, and 2 were unimproved. Davies³ produced a unilateral phrenic paralysis in 25 cases of basal bronchiectasis. Ten (right side) were not improved, and thoracoplasty was proposed; 5 (left side) obtained symptomatic relief and coughed less, but their condition was reported as worse eighteen months later; 1 (right side) was completely free of all symptoms for two years and then had recurrence with hæmoptysis and death. In a recent report¹⁵ he states that of 5 cases in which

* From the Department of Surgery of the University of Toronto and the Toronto General Hospital. Based on the work of the author and his senior colleague, Dr. N. S. Shenstone. Read at the combined meeting of the Canadian and Ontario Medical Associations at Toronto, June 24, 1932, as the surgical contribution to a symposium on bronchiectasis.

Table I.—RESULTS OF SEVEN CASES

CASE	SEX	AGE	LOCATION	DURATION	ADMISSION	OPERATION
1. H. H.	M.	32	R.L., R.M.	1 year ..	June 21/28 Feb. 27/29	Phrenic neurectomy Oct. 9/23 Multiple-stage extrapleural thoracoplasty between March 6 and April 19/29
2. A. M.	M.	27	L.U. and L.	1 year, 3 months	June 18/30 Sept. 19/31	Multiple-stage extrapleural thoracoplasty between July 30 and Oct. 21/30
3. A. W.	F.	24	L.U. and L.	Well until 10 days before admission 13 months ..	Feb. 15/30 March 12/31	Thoracotomy Feb. 25/30 Multiple-stage extrapleural thoracoplasty between April 1 and May 20/31
4. J. C.	M.	43	L.U.* ..	1 year, 8 months	Jan. 26/32	Drainage of abscess Feb. 11/32 Thoracoplasty 1-4 posterior April 2/32 5-9 posterior April 23/32
5. H. H.	M.	50	R.U. ..	12 years ..	July 2/32	Upper right posterior thoracoplasty July 22/30
6. H. W.	M.	43	L.U. and L.	16 years ..	June 6/32	Multiple-stage thoracoplasty July 8/32 to Aug. 30/32
7. R. D.	M.	24	L.U. and L.	8 years ..	Aug. 8/32	Multiple-stage thoracoplasty July 11/32 to Oct. 13/32

* Bronchiectasis occurred as complication of lung abscess.

SURGICAL TREATMENT OF BRONCHIECTASIS 259

BRONCHIECTASIS TREATED BY THORACOPLASTY.

ANESTHESIA	DISCHARGE OR DEATH	COMPLICATIONS	RESULTS
Costal nerve-block	Jan. 28/29 May 21/29	Septic pneumonia left lower lobe with large abscess	Became worse during period of operative treatment. Had extension to left lower lobe with formation of abscess. At autopsy right lung fibrous throughout and contained almost no pus
Costal or intercostal nerve-block for posterior. Gas and oxygen anterior	Nov. 9/30 Oct. 7/31	Carcinoma of bronchus ..	In course of thoracoplastics sputum gradually decreased from 12 to 14 oz. daily to a maximum of 1 oz. daily. Following discharge Nov. 9/30 health improved for 7 or 8 months and then began to suffer from attacks of dyspnoea. Upon re-admission Sept. 19/31 found to be still in fair general health with little sputum. Died. Autopsy showed carcinoma the size of a walnut at entrance to left primary bronchus
Costal for lower. Gas and oxygen for upper anterior	Aug. 1/30 Aug. 8/31	Empyema Nil	Patient admitted with widespread bronchopneumonia left side. Empyema occurred which was drained. Went on to a widespread bronchiectasis. Transferred to sanatorium, where a gain of 20 lb. in weight occurred but sputum remained unchanged Patient developed a small amount of bronchitis of opposite side. Stood operations well, but there was little decrease in sputum. Not much improved When heard from several months later had gained somewhat in weight and improved in general health. Sputum only slightly decreased
Costal and oxygen ..	June 13/32		Upon discharge from hospital general health good. Sputum consisted of a little clear mucus only. Drainage wound almost healed. No discharge March 1/33. No cough, no sputum
Costal and oxygen ..	July 29/30	Bronchopneumonia left side	Post-operative course at first fairly satisfactory, temperature to 101°. Died July 29. Autopsy showed an extensive sacular bronchiectasis of the right upper lobe, adherent pleura right side. Bronchopneumonia on left side apparent cause of death
Costal, gas and oxygen, local	Sept. 12/32	Mild phlebitis left femoral	March 20/34. Looks well. Cough and sputum still troublesome, but sputum 2 to 4 oz. daily as compared with maximum of 20 oz. daily before operation, and less foul
Costal, gas and oxygen, local	Nov. 21/32	On Oct. 25/32 developed a widespread bronchopneumonia in the right lower and middle lobes, gradually recovering from this. On discharge there were only a few rales at the right base. Sputum 4 to 8 oz. daily and foul	March 16/33. Has gained 30 lb. since leaving hospital. Sputum, about 20 oz. before operation, now about 4 oz. daily, but still very foul. Has had no further pneumonia

R.L. = Right lower. R.U. = Right upper. R.M. = Right middle. L.U. and L.L. = Left upper and lower.

the operation was done in the hope that it might be self-sufficient, 4 gained no benefit though the fifth was relieved of symptoms for two years. In two other cases in which the evulsion was done as a preliminary to thoracoplasty, the major operation had been postponed indefinitely on account of the improvement resulting from the hemidiaphragmatic paralysis. In our experience the relief obtained by paralysis of the diaphragm has not been worth while.

Pneumothorax has not been used by us in the treatment of the disease. Several of our cases had been treated in this manner, however. In some the sputum had been decreased temporarily, in others no noticeable improvement had occurred. Where adhesions do not prevent its use we believe it should be employed pre-operatively as an aid in emptying the dilated bronchi of their infective contents and perhaps lessening the danger of pneumonia. We consider, however, that, except perhaps in early cases in children, artificial pneumothorax should be used only as a pre-operative measure, since, if the air is allowed to absorb, the subsequent development of adhesions renders the operative procedure more difficult: such adhesions also inhibit the proper expansion of the upper lobe, preventing the obliteration of the cavity left after operation and increasing the likelihood of a bronchial fistula.

The number of cases which we have treated by *thoracoplasty* is too few to form the basis of an opinion. The results obtained may be seen in *Table I*. *Case 1* was one of the rapidly progressive type. Although he died shortly after the completion of the collapse, the condition of the compressed lung at autopsy suggested that, had the infection of the opposite lung not occurred, marked improvement might have been expected. *Case 5* also had an extension to the good side. *Case 2* should, of course, never have been operated upon, and illustrates the necessity for a bronchoscopic examination. The decrease in sputum and improvement in general health were, however, sufficient to have made the operation well worth while. *Case 3* was improved, but not to the extent that might have been expected. *Case 4* is well. *Cases 6 and 7* are moderately improved at present, but too a short time has elapsed to permit a final judgement.

In 1931 Hedblom¹ reported that of 14 cases that had had a thoracoplasty before 1926, 3 had remained symptom-free, 7 raised from 10 to 20 per cent less sputum: 6 of these were working regularly, 4 who were improved died from ten months to two years after operation—one of actinomycosis. Of 18 cases operated upon since 1926, 4 died (one of brain abscess, one of pulmonary gangrene, and two of pneumonia). The remaining 14 were all improved, with sputum reduced from 60 to 90 per cent; 2 died following a secondary lobectomy. In view of the results that may be obtained by lobectomy it would seem that extrapleural thoracoplasty should be reserved for those cases of unilateral bronchiectasis in which the area of involved lung is too great to be removed completely. The degree of collapse required is much greater than in pulmonary tuberculosis. Because of the large amount of sputum, local or spinal anaesthesia is preferable, and the operation should be done in multiple stages, usually at least four.

Drainage.—The drainage of one or more of the large abscess pockets would appear to have been used extensively by Sauerbruch. He recommends

its employment⁴ and states that it often results in considerable alleviation of symptoms but never in cure. The pathology of the condition makes it clear that little could be expected from simple drainage. We have not attempted to treat bronchiectasis *per se* by drainage, but have consistently failed to obtain a cure by drainage alone in those cases of chronic lung abscess in which a secondary bronchiectasis had developed.

LOBECTOMY.

That lobectomy would be the ideal method of treatment of localized bronchiectasis has been recognized by most writers on the subject. Surgeons have been loath to advocate it because of the tremendous mortality associated. Lilienthal reported a personal mortality of 47 per cent. Coryllos⁵ in 1930 collected 87 cases operated upon by eleven different surgeons, with 48 deaths—a mortality of 53.1 per cent. Of the surviving cases, 38 were cured by operation and 1 was improved. Coryllos reported 2 of his own cases, 1 cured without a fistula, 1 cured apart from the presence of a fistula. He advocated a preliminary graded thoracoplasty followed by lobectomy in one or two stages; one lobe was removed with a cautery. Whittemore⁶ described a method in 1927 in which under gas and oxygen anaesthesia a sufficient number of ribs were removed to permit of the diseased lobe being freed and brought to the surface, where it was firmly fixed to the chest wall by deep sutures of silk and chromic catgut. In about ten days the lung tissue became necrotic, and in four to five weeks it sloughed away, leaving a clean granulating wound, which was lightly packed until it closed. The cases were in hospital for an average of six to eight weeks, and the wound required from three to four months to close. He did not do a preliminary thoracoplasty, but suggested that it might be wise to do so. He reported 6 cases, 3 of which were healed without fistula: one was well but had a persistent fistula at the end of one year and three months; one was still in hospital; and one died on the tenth day from pneumonia on the good side.

Robinson^{7, 8} described a method of two-stage lobectomy. At the first sitting the overlying ribs, periosteum, and intercostal bundles were removed. At the second stage the lobe was freed, clamped at the pedicle, and excised. Individual vessels were ligated, and finally a mass ligature of silk or kangaroo tendon was placed on the pedicle. The cavity was packed and allowed to granulate. He reported 5 cases with 1 death⁸, and stated that the resulting bronchial fistulae were easily closed by skin- and fat-grafts. In his other paper he reported 7 cases with 3 deaths. Archibald¹⁰ successfully performed a two-stage lobectomy in 1924.

Graham⁹ advocated the cautery excision of the diseased lung. Part of the overlying chest wall was first removed and the wound packed widely open. The diseased tissue was destroyed later in multiple stages with a cautery iron. He reported 20 cases, of which 4 (20 per cent) were completely healed, 6 (30 per cent) were free of symptoms but had bronchial fistulae, 3 (15 per cent) showed marked improvement but still had some cough, 2 were still in process of treatment, 1 had not been heard from, 4 (20 per cent) were dead.

Hitzrot¹¹ reported a successful one-stage lobectomy in 1920. The chest was opened through an intercostal incision in the seventh interspace; the lobe was freed from dense adhesions and removed. The intercostal wound was closed completely and drainage established by the Kenyon method through an intercostal stab-wound. A bronchial fistula developed, but closed spontaneously. A complete cure was accomplished. McDowell¹² reported a single successful case operated upon by Tudor Edwards in 1930. The chest was closed tightly and the pleural effusion, which remained sterile, aspirated upon ten occasions. The patient, a boy of 16, became completely well. That the effusion would remain sterile in many cases could not be expected.

TECHNIQUE.

The technique which we have developed is a modification of that employed by Brunn¹³. It was described by Shenstone in a paper given before the conjoined meeting of the British and Canadian Medical Associations in Winnipeg, on Aug. 27, 1930.

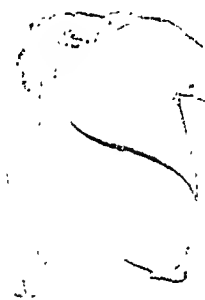


FIG. 182.—Position of incision in relation to inferior angle of scapula.

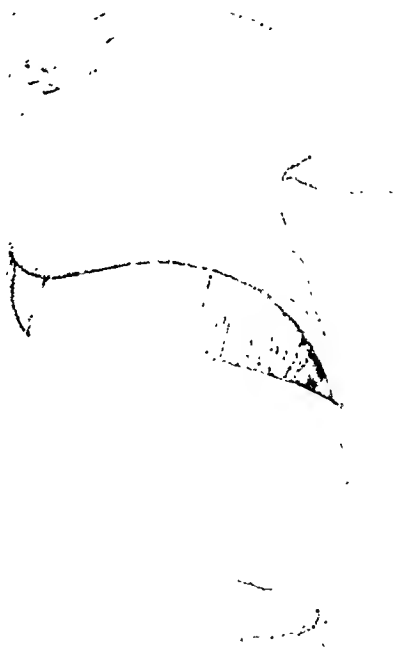


FIG. 183.—Trapezius, latissimus dorsi, and serratus magnus exposed.

A long incision is made in the general direction of the ribs, passing just below the scapula and extending from a point a few inches above and behind the angle of that bone to the costal cartilages in front (Figs. 182, 183).

The underlying muscles are severed in the same direction (*Fig. 184*), exposing the ribs and intercostal spaces. We have usually chosen the sixth interspace as our portal of entry into the chest, though the fifth or seventh appears to be equally satisfactory. The subsequent exposure is much enlarged by



FIG. 184.—Division of trapezius and latissimus dorsi muscles, exposing erector spinae and serratus anterior muscles, 6th, 7th, and 8th ribs.

dividing the ribs adjacent to the selected interspace as near their vertebral ends as possible, and this we have done in each case before entering the thorax (*Fig. 185*). For this purpose the lateral margin of the erector spinae is dissected medially, and the tip of a straight bone-cutting forceps inserted close to the rib, which is severed without prior elevation of the periosteum.

In the earlier cases the intercostal muscles were divided carefully and air allowed to enter slowly by plugging the opening intermittently with the tip of the finger (*Fig. 186*). We now feel that cases with a free pleura should have a preliminary pneumothorax. If this has been done, no special precautions are necessary. The intercostals with the underlying pleura may be divided safely from the costal cartilage to the angle of the ribs and the

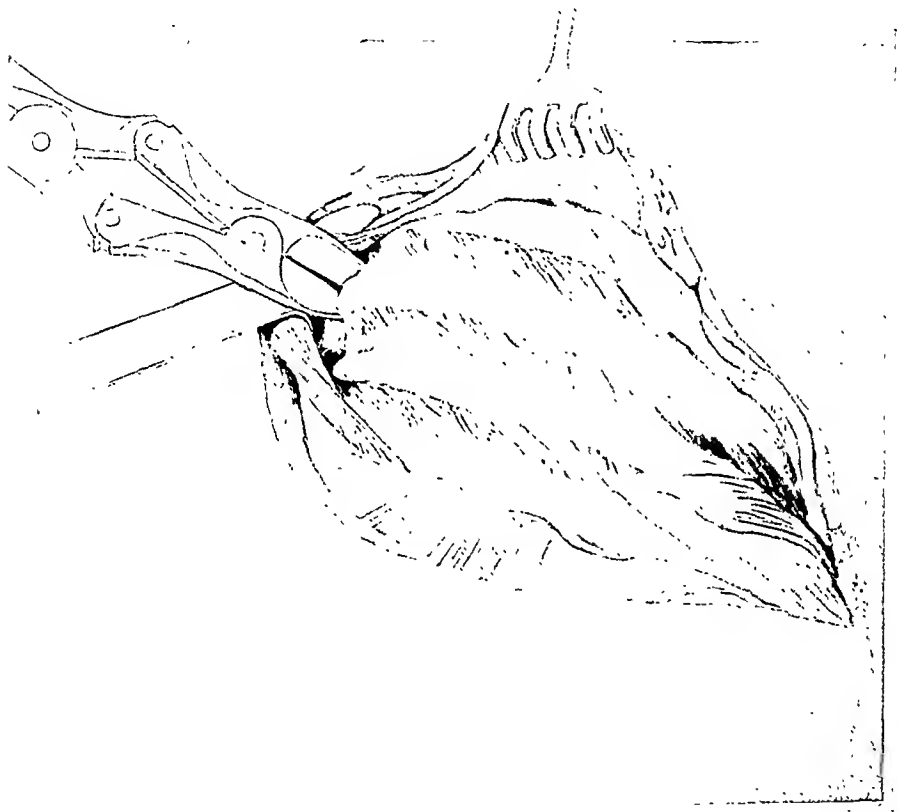


FIG. 185.—Elevation and retraction of the lateral border of the erector spinæ muscle, permitting the section of the 6th and 7th ribs near their vertebral extremity.

wound spread widely with self-retaining retractors (*Fig. 187*). An enormous exposure is obtained in this way.

Adhesions are variable: in our experience they are always present in the interlobar fissure, and usually so between the lower lobe and the diaphragm. In several of our cases the pleural space was obliterated. Adhesions are separated by a combination of sharp and blunt dissection, and great care must be taken to avoid wounding the lung.

When the diseased lobe has been freed completely a snare of heavy cord is passed around it as near as possible to the mediastinum, and the loop drawn tight in the instrument designed for the purpose (*Fig. 188*). We believe

it to be a better method of controlling the pedicle than any other we have seen described, and nothing has given us such a feeling of comfort and safety in this operation as this simple device. Without the devitalization of the tissue, it controls the blood-supply, obstructs the bronchi, provides a solid support during the suture of the pedicle, and, in addition, can be used in limiting mediastinal movement during the operation.

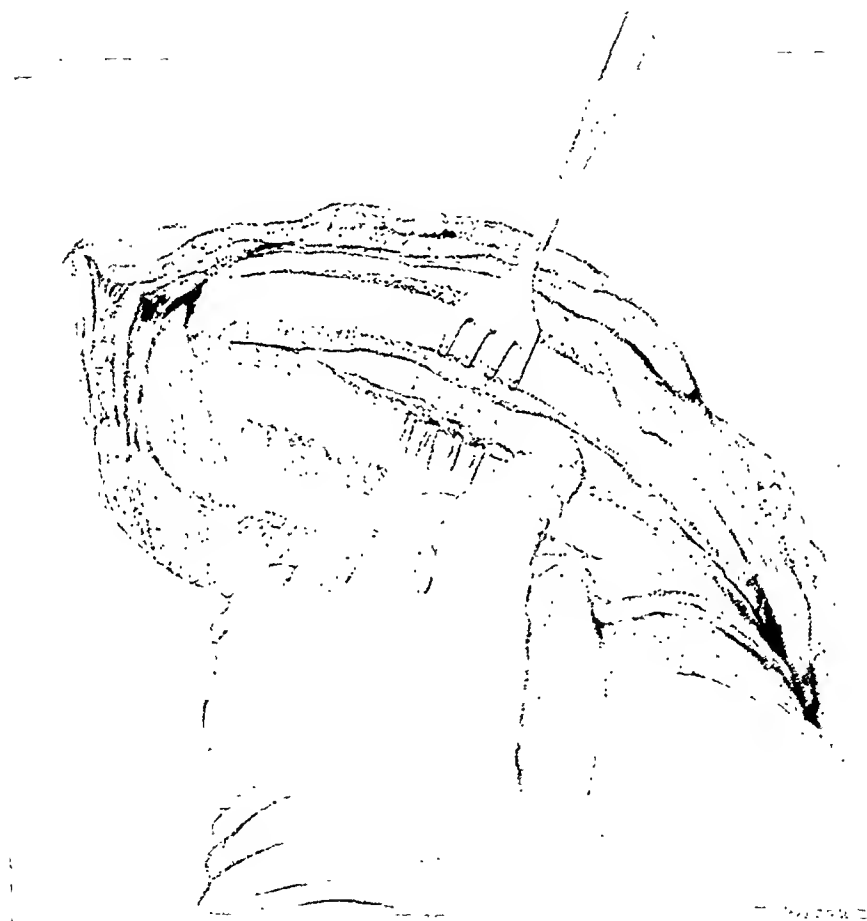


FIG. 186.—Intercostal muscles divided: 6th and 7th ribs retracted. Tip of finger placed over opening in pleura to ensure slow entry of air into pleura. (After Brunn.)

For the purpose of preventing the escape of infected material during the section of the lung, a second snare is placed about $1\frac{1}{2}$ in. distal to the first in those cases in which the diseased lobe is sufficiently voluminous (Fig. 189). When the lobe to be resected has become very small as a result of extensive fibrosis or collapse, this second snare has been found to interfere

with the making of a properly excavated pedicle and its use has been discontinued. The general pleural cavity is then protected by moist packs and the lobe cut away, leaving a pedicle $\frac{3}{4}$ in. in length. The section is best accomplished with curved scissors, and the cut surface of the pedicle should be somewhat excavated to assist in the subsequent closure (*Figs. 190-193*).

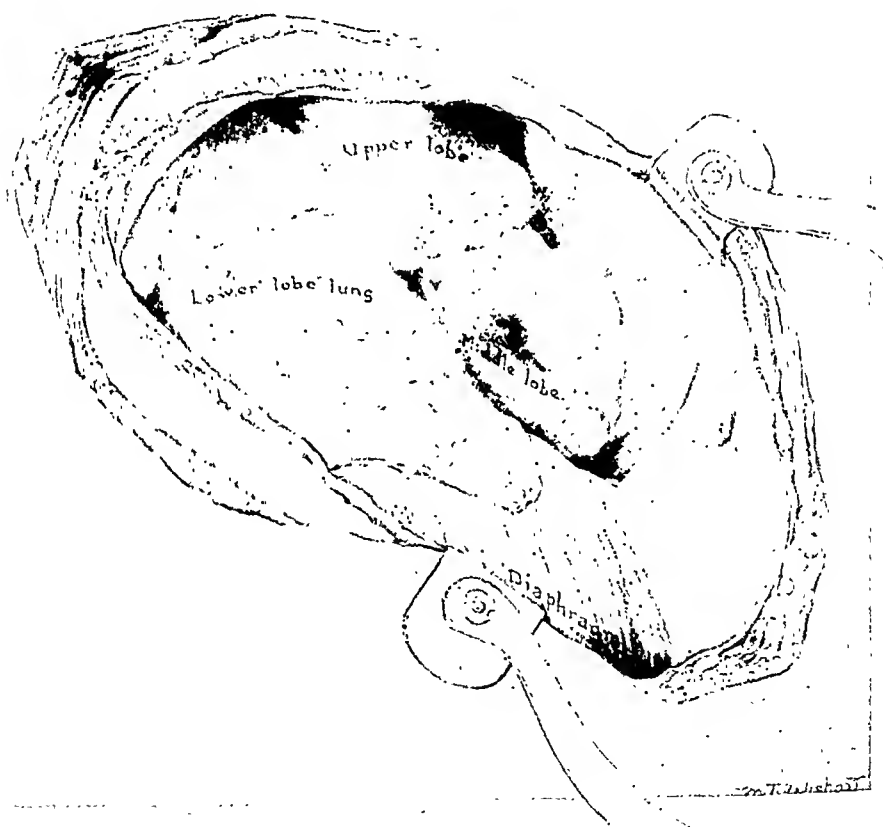


FIG. 187.—Exposure obtained in a case without pleural adhesions.

Frequently one or more bronchi project above the surface: these should be trimmed away, but care should be taken that a bronchus with a bronchial artery does not slip back out of the snare, as this may result in the vessel being missed by the running suture and subsequent hæmorrhage into the bronchus. The resultant bare area is painted with acriflavine (1-1000).

Obvious vessels are caught with forceps and ligated, and a running suture of catgut is introduced across the pedicle but not including its pleural margin. The snare is then slightly released and any oozing areas can be included in the suture before it is tied. A further running catgut stitch closes over the pleural margins, an endeavour being made to invert the edges in the process. The snare is then removed and the remaining lobe carefully examined for any small tears overlooked earlier.

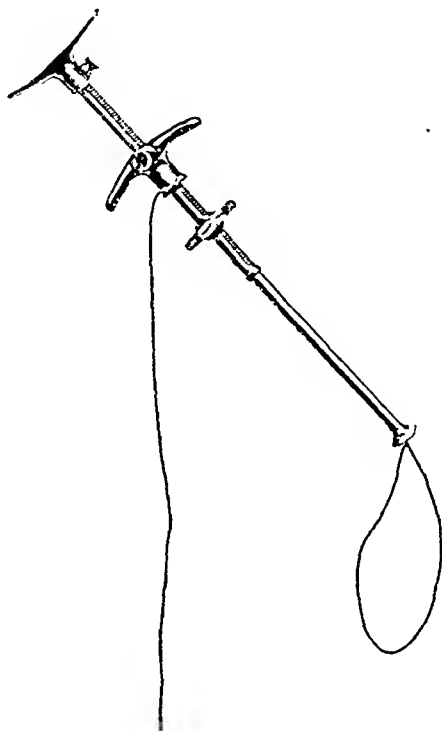


FIG. 188.—Special tourniquet designed to control the pedicle.

It is important to have decided from lipiodol injection before operation whether or not there is an involvement of the middle lobe on the right or the lingual portion of the upper lobe on the left side. We have found that inspection and palpation of the lung through the open chest cannot be relied upon. The middle lobe is occasionally sufficiently pedunculated to be freed and removed along with the lower. More often it must be dealt with separately, and is then removed with a snare in the same way as the lower lobe, or, where it cannot be freed to a definite pedicle, it must be clamped and removed in the same manner as a portion of the upper lobe.

When it is desired to remove part of a lobe, two crushing clamps are applied proximal to the diseased area and the lung sectioned between them.

The cut surface is painted with acriflavine. A running suture of chromic catgut is then placed over the forceps, the forceps removed, and the suture pulled taut. The bare area is covered in with a continuous Lembert suture. A small incision is then made in the ninth interspace in front of the mid-axillary line through which the end of a long tube of about 32F calibre is drawn. The fenestrated end of this tube is placed about 1 in. away from



FIG. 189.—Tourniquets in place. Exposed surfaces protected with gauze pads.

the pedicle of the resected lung and maintained in place by a plain catgut suture inserted into the summit of the diaphragm (*Fig. 194*).

The operation wound is closed in layers. Interrupted chromicized catgut sutures enclosing the adjacent ribs bring the edges of the long rent in the intercostals easily and accurately together (*Fig. 195*). In our experience the inclusion of the nerves has not caused any lasting pressure neuritis. The

muscles are approximated by a continuous suture of chromicized catgut and the skin by an ordinary dermal suture.

As soon as the wound is closed, the distal end of the drainage tube is placed under the surface of a normal saline solution contained in a bottle, maintained at least 2 ft. below the level of the chest. The patient is then invited to cough. This act expresses the air from the pleural cavity, permits of the rapid expansion of the lung, and prevents mediastinal flapping. At the end of twenty-four hours it has been our custom to attach a continuous

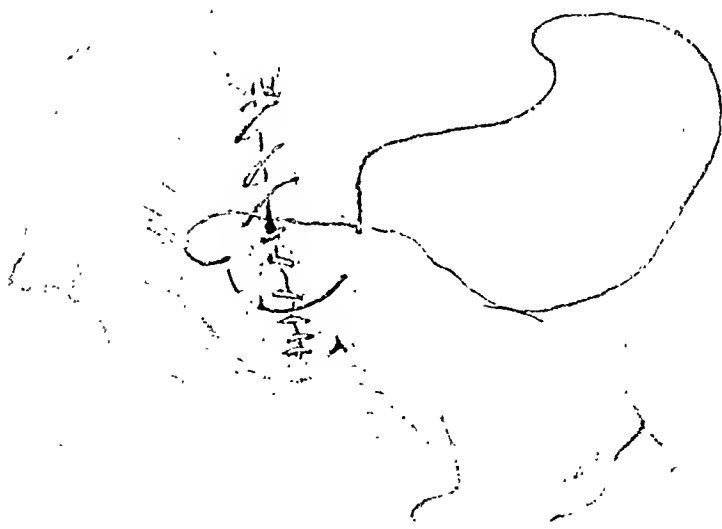
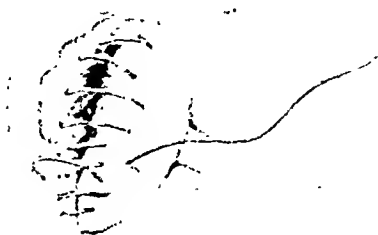


FIGS. 190, 191.—Ablation of lobe, leaving pedicle with concave surface.

siphon drainage to this tube, using a column of about 18 in. of water. The earlier cases were done under intercostal nerve-block, but spinal anaesthesia has been found much more satisfactory.

The use of the snare has made it easy to deal with the pedicle. There is no loss of blood, no escape of air, and much less spread of infected material. The flapping of the mediastinum may be entirely controlled. Apart from the fact that the placing of sutures is made so much easier, the patient experiences great comfort from this support and coughs much less frequently.

Under spinal anæsthesia there is a minimum of shock and it has been possible to proceed without hurry. The patient is able to cough and clear a considerable amount of the material expressed into the trachea. Flooding of the



FIGS. 192, 193.—Closure of pedicle and its subsequent suture to upper lobe.

opposite lung is not avoided entirely, however, and the occurrence of pneumonia in the good lung remains the greatest hazard. One case developed a massive collapse of the opposite lower lobe and subsequently

a bronchiectasis: this was probably due to the aspiration of tenacious sputum into that bronchus.

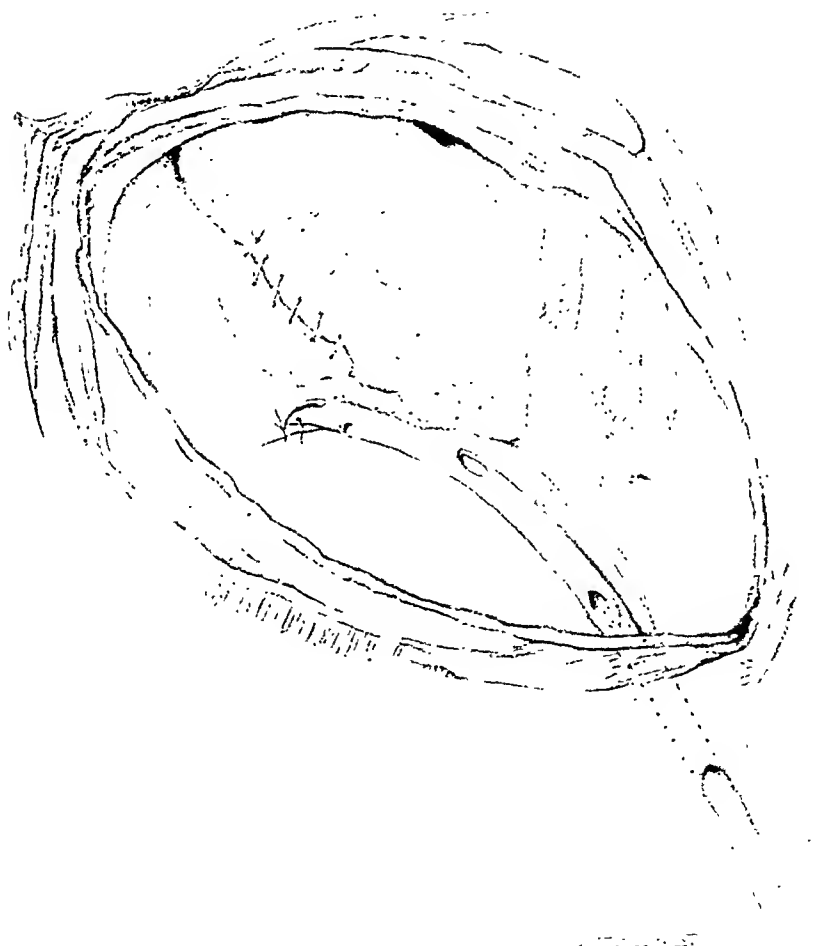


FIG. 194.—Drainage tube in place.

The patient is placed in bed in the Fowler position. Post-operative respiratory embarrassment and cyanosis is variable: in most cases it is slight, but occasionally is very considerable. It is often necessary to administer

oxygen until the pressures have been readjusted, and no patient should be operated upon unless an oxygen tent or oxygen chamber is available. Repeated hypodermic injections of atropine $\frac{1}{100}$ gr. help to control the œdema, but this does not seem to be nearly so effective as the administration of oxygen. Fairly frequent changes of position should be insisted upon. The

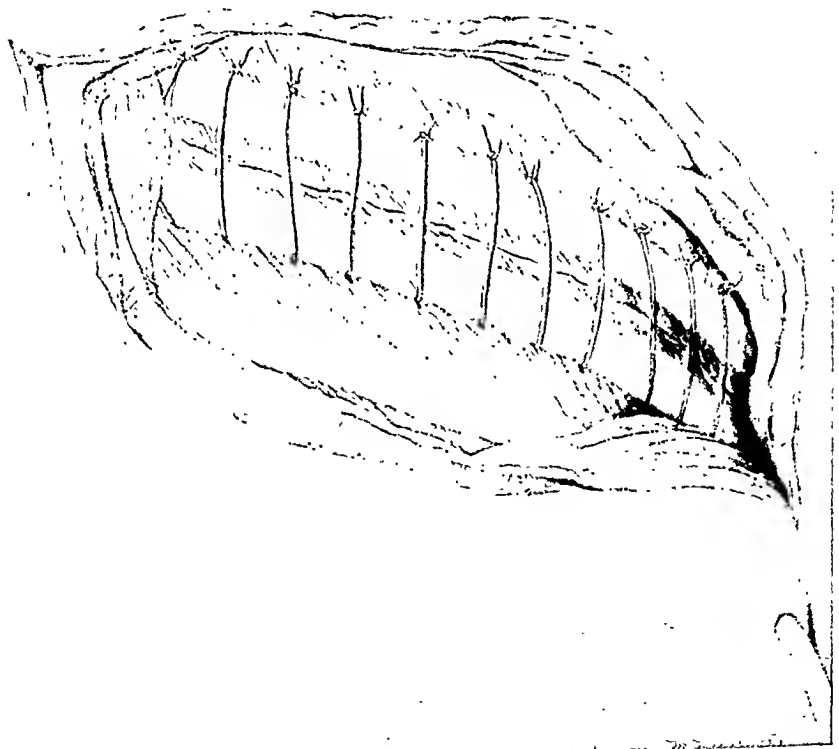


FIG. 195.—Method of closure: medial suture includes erector spine muscle.

patient should be encouraged to cough and thus clear bronchial secretions. Morphine should be administered in $\frac{1}{6}$ -gr. doses sufficiently frequently to ensure reasonable comfort and to permit of coughing without too much pain. For the first twenty-four hours there is a considerable escape of blood and serum. If the upper lobe is free from adhesions, it is displaced downward and quickly becomes adherent to the elevated dome of the diaphragm.

It is this application of the upper lobe to the diaphragm as well as the method of closure of the stump that prevents the formation of a bronchial fistula, and we have noted that if the descent of the upper lobe is prevented by pleural adhesions, a fistula is more likely to occur. In many of our cases there has occurred a temporary pneumothorax in the upper pleura in which a variable amount of fluid has been present. Both air and fluid may be absorbed rapidly, the lung expanding to fill the space. In two of the cases this condition was accompanied by infection requiring drainage through intercostal stab-wounds. In one of these rapid healing took place; in the other a partial thoracoplasty was required for closure of a persistent cavity. More recent experience has convinced us that collections of fluid above the upper lobe should be drained as soon as recognized. An X-ray should be made with the patient in the sitting position within the first two or three days following the operation, as it is not always possible to interpret correctly the clinical findings. If drainage is established promptly, infection may be prevented, any embarrassment to respiration is removed, the lung is allowed to expand more rapidly, and the cavity is much more likely to close spontaneously.

Two cases developed increasing fever for which no cause could be discovered up to the tenth day post-operatively, at which time they suddenly coughed up several ounces of pus. The X-ray of one such case showed a definite cavity with a fluid level about the stump of the resected lung. In both cases cough and sputum ceased at the end of four to six weeks. They are both well and apparently cured. This complication prolongs convalescence, but since the abscess is efficiently drained into a main bronchus it would appear that spontaneous recovery could be anticipated.

SUMMARY OF RESULTS.

An analysis of *Table II* reveals the following points of interest.

Of 17 cases operated upon, 16 were done by the one-stage method. Of these 16 cases, 6 were cured, 3 were improved, 2 unimproved, and 5 died. In one case (*Case 5*) improvement only (and not cure) occurred from failure to recognize an involvement of the lower portion of the upper lobe on the same side; in another (*Case 9*) owing to the presence before operation or possibly the development after operation of some disease in the opposite lobe. One (*Case 14*) developed a post-operative collapse of the previously healthy opposite lower lobe, in which there is now a well-developed bronchiectasis. The two unimproved cases were more extensive than was realized. Complete post-mortem examinations were obtained upon the 5 cases that died. Two deaths (*Cases 4* and *8*) were found to have been due to bronchopneumonia in the previously normal lung of the opposite side. *Case 16* had a large amount of sputum, but lipiodol injection seemed to demonstrate that the bronchiectasis was confined to the left lower and lower part of the left upper lobe. Autopsy showed that death had been due to bronchopneumonia, empyema, and suppurative pericarditis; the remaining portion of the left upper lobe and right lower lobe were bronchiectatic. A cavity the size of a hen's egg in the upper lobe had failed to fill with lipiodol. This was very disturbing, and demonstrated that even the lipiodol injections cannot be depended upon

entirely for localizing the disease. Had the extent of the lesion been appreciated, one would not have thought of attempting even a thoracoplasty. There was no evidence that the pericardium had been injured at operation and no apparent reason for the occurrence of the pericarditis. The fourth case (*Case 11*) died of secondary hæmorrhage from a perforating branch of the internal mammary artery eight months after the primary operation and three weeks after a persistent bronchial fistula had been closed, apparently successfully, by a plastic operation. This death should be looked upon, probably, as an unfortunate accident, since the diseased lung had been removed entirely, but is a reminder of the fact that no case in which a fistula is still present can be regarded as out of danger. The fifth case died of a brain abscess fifty-two days after operation.

Of the 16 cases, 6 developed bronchial fistulæ. Three of these were temporary only and closed spontaneously, one required an operation for closure (*Case 11*), and in two (*Cases 16, 17*) the patient failed to survive. When the upper lobe is freely movable and becomes adherent to the dome of the diaphragm over the drainage tube, the fistula, should one form, consists of a long, narrow tract and closes readily. When, however, an empyema forms between the upper lobe and the diaphragm and the large bronchus opens directly into this, spontaneous closure is not usual and a plastic operation may be necessary later. It is interesting that of 12 left-sided cases operated upon, only 3 developed fistulæ; of these, the one in the patient that survived closed readily. On the other hand, of 4 right-sided cases operated upon, 3 developed fistulæ, one of which was closed by operation.

Of the 16 cases, 4 developed more or less extensive empyemata. Two of these were above the upper lobe; one closed readily following intercostal drainage; one was closed by thoracoplasty. Two formed between the upper lobe and diaphragm; the one in the patient that survived was closed by thoracoplasty. (*Table II.*)

Massive collapse of the opposite lobe occurred once only, but proved a serious complication, since a bronchiectasis subsequently developed.

It is essential, before considering possible therapeutic measures, that the location and the extent of the disease be determined as accurately as possible. The usual physical examination of the chest and ordinary radiographs do not give the definite evidence that is necessary, and, in all cases, most careful injections of the bronchial tree with lipiodol must be made, especial attention being given to the condition of both lower lobes, the inferior part (lingual lobule) of the left upper, and the right middle lobes. Even with the utmost care in the injections an occasional diseased bronchus is missed.

These examinations having been carried out, we have accepted for surgical consideration cases limited to one lower lobe with or without involvement of the lingual lobule on the left side or the corresponding middle lobe on the right.

We have considered the operation justifiable in those patients whose sputum was large in amount or foul, in those who had suffered numerous pulmonary hæmorrhages whether or not they had sufficient sputum to be

Table II.—RESULTS OF LOBECTOMY IN BRONCHIECTASIS.

CASE	SEX	AGE	DATE OF ADMISSION	DATE OF OPERATION	CHARACTER OF OPERATION	DATE OF DISCHARGE OR DEATH	POST-OPERATIVE DAYS IN HOSPITAL	TEMPORARY PISTILA	COMPLICATIONS	RESULT
Two-stage Lobectomy—										
1. M. S.	F.	34	May 22/28	Jan. 22/29 Mar. 6/29	R.L. . .	Mar. 11/29	5 died	No	Bronchopneumonia	Died
One-stage Lobectomies—										
2. M. R.	F.	19	May 3/29	May 4/29	R.L. and R.M.	June 9/29	36	Yes	Osteomyelitis of rib	Cured
3. A. D.	M.	47	Nov. 24/29	Nov. 30/29	L.L. . .	Dec. 23/29	23	No	—	Cured
4. C. C.	F.	20	Aug. 20/29	Dec. 18/29	R.L. and R.M.	Dec. 23/29	5 died	Yes	Bronchopneumonia	Died
5. R. L.	F.	26	Nov. 5/29	Feb. 21/30	L.L. . .	May 29/30	99	No	Bronchopneumonia	Improved
6. V. W.	F.	23	Feb. 17/30	Mar. 26/30	L.L. and part of L.U.	June 30/30	96	No	Empyema (U) drainage	Improved
7. J. S.	M.	30	Feb. 12/30 June 30/30	July 2/30	L.L. . .	Apr. 17/30 Oct. 29/30	89	No	Empyema (U) drainage and thoracoplasty . . .	Unimproved Died
8. L. S.	M.	52	Aug. 31/30	Oct. 8/30	R.L. . .	Oct. 14/30	6 died	No	Bronchopneumonia	Unimproved
9. D. McG.	F.	14	Oct. 24/30	Nov. 1/30	L.L. . .	Dec. 17/30	46	No	Pleurisy and neutro bronchitis . .	Improved
10. K. F.	F.	21	Jan. 21/31	Feb. 11/31	L.L. . .	May 5/31	83	No	Secondary infection of wound	Improved
11. F. L.	M.	22	Jan. 28/31	Feb. 27/31	R.M. and R.L.	Oct. 30/31	245 died	Yes	Empyema and secondary hemorrhage	Died
12. B. O'B.	F.	24	Mar. 25/31	Apr. 1/31	L.L. . .	June 18/31	78	No	—	Unimproved
13. M. K.	F.	20	July 28/31	Aug. 19/31	L.L. and part of L.U.	Oct. 1/31	43	No	—	Cured
14. M. C. S.	F.	30	Aug. 14/31	Aug. 19/31	L.L. . .	Oct. 31/31	63	No	Massive collapse of right lower lobe . .	Improved
15. G. H.	F.	32	Nov. 23/31	Dec. 9/31	L.L. . .	Jan. 21/32	43	Yes	—	Cured
16. J. T.	M.	24	Apr. 19/32	Apr. 27/32	L.L. and part of L.U.	May 27/32	38 died	Yes	Bronchopneumonia, pericarditis, and empyema . .	Died
17. M. L.	F.	17	Apr. 26/32	Apr. 30/32	L.L. . .	June 21/32	52 died	Yes	Brain abscess . .	Died

R.L. = Right lower, R.M. = Right middle, L.L. = Left lower, L.U. = Left upper.

in itself significant, and in those in whom the disease appeared to be definitely progressive.

A review of our series has demonstrated that cases with persistent fever and those recently recovered from exacerbations of the disease are unsuitable for operation. We feel that a considerable afebrile interval should elapse before lobectomy is advised in these cases, and that the procedure is probably safer if carried out during the warmer weather of the year. The post-operative septic pneumonias following operations where these obvious precautions are not taken are of a peculiarly serious type and have been the immediate cause of death in 40 per cent of our fatal cases. Patients having a large amount of sputum should carry out postural drainage for some time before operation, and we prefer to operate upon them in the later, rather than in the early, part of the morning. If the sputum is foul, and particularly if numbers of spirochaetes are demonstrable, it is possible that one or two administrations of one of the intravenous preparations of arsenic should be given before operation.

It is a pleasure to acknowledge my indebtedness to Dr. Shennstone for the privilege of reporting this work and for much helpful criticism in the preparation of the paper, and to Miss M. T. Wishart for the patience and care with which she has prepared the illustrations.

REFERENCES.

- ¹ HEDBLUM, C. A., "Pathogenesis, Diagnosis and Treatment of Bronchiectasis", *Surg. Gynecol. and Obst.*, 1931, lii, 406.
- ² OAKLEY, W. G., "Indications for Phrenic Evulsion in Bronchiectasis", *Brit. Med. Jour.*, 1931, ii, 378.
- ³ DAVIES, H. M., "An Endeavour to Assess the Value of Hemidiaphragmatic Paralysis by Evulsion of the Phrenic Nerve in the Treatment of Pulmonary Tuberculosis and Bronchiectasis: A Review of 105 Cases", *Tubercle*, 1927-8, ix, 205.
- ⁴ SAUERBRUCH, E. F., *Die Chirurgie der Brustorgane*, 564-590.
- ⁵ CORYLLOS, P. N., "Treatment of Bronchiectasis—Multiple Stage Lobectomy: Report of Two Cases", *Arch. of Surg.*, 1930, xx, 767.
- ⁶ WHITTEMORE, W., "The Treatment of such Cases of Chronic Suppurative Bronchiectasis as are Limited to one Lobe of the Lung", *Ann. of Surg.*, 1927, Aug., 219.
- ⁷ ROBINSON, S., "The Resection of Lobes of the Lung", *Jour. Amer. Med. Assoc.*, 1917, Aug. 4, 355.
- ⁸ ROBINSON, S., "The Surgery of Bronchiectasis, including a Report of Five Complete Restorations", *Surg. Gynecol. and Obst.*, 1917, xxiv, 194.
- ⁹ GRAHAM, E. A., "Cautery Pneumonectomy in Thoracic Suppuration of the Lung, a Report of Twenty Cases", *Arch. of Surg.*, 1925, x, 392.
- ¹⁰ ARCHIBALD, E., "The Surgical Treatment of Bronchiectasis", *Canad. Med. Assoc. Jour.*, 1924, March, 197.
- ¹¹ HITZROT, J. M., "Lobectomy for Bronchiectasis", *Ann. of Surg.*, 1920, June, 785.
- ¹² McDOWELL (for TUDOR EDWARDS), "Treatment of Bronchiectasis by Lobectomy", *Trans. Med. Soc. of Lond.*, 1930, 53.
- ¹³ BRUNN, H., "Surgical Principles underlying One-stage Lobectomy", *Arch. of Surg.*, 1929, Jan., 180.
- ¹⁴ SHENSTONE, N. S., and JAMES, R. M., "Experiences in Pulmonary Lobectomy", *Canad. Med. Assoc. Jour.*, 1932, Aug.
- ¹⁵ DAVIES, H. M., *Surgery of the Lung and Pleura*, 1930.
- ¹⁶ LILIENTHAL, *Thoracic Surgery*, 1925, ii.
- ¹⁷ FINDLAY, L., and GRAHAM, S., "Prognosis in Bronchiectasis", *Arch. of Dis. Child.*, 1931, Feb., 1.

PULMONARY LOBECTOMY.*
TECHNIQUE AND REPORT OF TEN CASES.

By J. E. H. ROBERTS,

SURGEON TO ST. BARTHOLOMEW'S HOSPITAL AND THE BROMPTON HOSPITAL FOR
CONSUMPTION AND DISEASES OF THE CHEST;

AND H. P. NELSON,

CHIEF ASSISTANT TO A SURGICAL UNIT, ST. BARTHOLOMEW'S HOSPITAL, AND ASSISTANT SURGEON
TO THE BROMPTON HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST.

THE technique of the operation we are about to describe is in all essential details that of Shenstone and Janes,¹ of Toronto. In 1931 one of us was in Toronto and had the advantage of seeing these surgeons perform their operation and of following the post-operative course; and this account is based on the experience of our first ten consecutive cases operated on at the Brompton and St. Bartholomew's Hospitals. We differ from them in some minor details of technique and have introduced several new instruments which we consider add to the facility of the operation.

Until quite recently lobectomy has had a mortality of about 50 per cent. The chief difficulties were a generalized infection of the pleural cavity, the closure of the pedicle after removal of the lobe, mediastinal infection, and mediastinal emphysema. On account of the danger of a total empyema many surgeons perform the operation in two or more stages. In the absence of adhesions Lilienthal² takes steps at the first stage to promote adhesions over the upper lobe either by rubbing the pleura with dry gauze or painting it with iodine. John Alexander³ does this as a routine at the first stage of his lobectomy operation. Some surgeons emphasize the danger of a large open pneumothorax in the absence of adhesions or rigidity of the mediastinum, but from our operative experience we are convinced that this is a false doctrine. During the one-stage operation the mediastinum certainly does shift towards the opposite side, but without any appreciable embarrassment to the patient's pulse or respiration. To overcome the difficulties of closure of the bronchial stump and pleural infection, Sauerbruch,⁴ Whittemore,⁵ John Alexander,³ Zaaier,⁶ and others advocate strangulation of the diseased lobe either by ligatures or a rubber band, following which the diseased lobe is drawn outwards to occlude the opening in the chest wall (exteriorization), and in the course of the next two weeks sloughs off. In order to obliterate the space left after the lobectomy, and also to promote adhesions, Harrington,⁷ Hedblom,⁸ and Coryllos⁹ advocate a thoracoplasty beforehand, but this is entirely unnecessary, because if the remaining lobe is free, it has no difficulty in expanding to fill the entire hemithorax.

*Submitted for publication, January, 1933.

In 1929 Harold Brunn¹⁰ pointed out that no serious infection of the pleura occurred in a one-stage operation if early expansion of the remaining lobe was encouraged. By closing the chest tightly and employing a negative pressure drainage he showed that the lobe became adherent before the discharge became purulent. Brunn's idea stimulated Shenstone and Janes to further work on the one-stage technique. Their operation differs from Brunn's only in the method of closing the pedicle. The one-stage operation in a free pleura is now shown to be at least as safe a procedure as the 'strangulation' operation, and has the advantage that there is no permanent diaphragmatic paralysis and that the remaining lobe is not crippled by dense adhesions and deformity of the chest wall.

ESSENTIAL PRINCIPLES OF THE OPERATION.

1. The entrance to the thorax through a long intercostal incision, with the result that there is no permanent deformity of the chest wall.

2. The freeing of adhesions not only round the diseased lobe but also over the costal surface of the remaining lobe so that it will be free to expand and fill the hemithorax after the operation.

3. The employment of two tourniquets on the pedicle of the lobe, the proximal one to control bleeding and the distal one to prevent escape of infected material while the lung is amputated between these two.

4. The closure of the stump by a continuous suture rather than by dissection and separate ligation of its component structures. This we feel is an essential point in prevention of mediastinal infection or emphysema.

5. Negative pressure drainage to remove any fluid and encourage the early expansion of the upper lobe, in order to obliterate all the dead space and so reduce the infected area to a small track.

PRE-OPERATIVE CONSIDERATIONS.

Indications.—As the object of this paper is to describe a technique, we do not wish to dwell at length on the indications or contra-indications for lobectomy. It is obviously an ideal treatment for a chronic disease which is confined to one only of the lobes of the lung. Bronchiectasis, either cystic, atelectatic, or acquired, is the condition for which the majority of operations are performed. The best results are naturally to be expected when the disease is strictly confined to one lobe, but we feel nevertheless that when there is advanced disease, say in one lower lobe, with a slight amount in the opposite base or middle lobe of the same side, one is still justified in removing the grossly diseased part. Bronchial carcinoma of the peripheral type with no X-ray or bronchoscopic evidence of mediastinal involvement is an indication for lobectomy, but primary tumours of this type are unfortunately rare. We feel that a chronic lung abscess or multiple abscesses confined to one lobe would probably be best treated by this operation, but have not yet had a suitable case.

Bronchoscopy and Postural Drainage.—When operating on patients who have much sputum there is considerable danger of bronchopneumonia in the opposite side, and this is one of the most potent causes of death in either

one-stage or two-stage lobectomy. Every effort must therefore be made beforehand to reduce the sputum to the minimum. The amount of sputum can be considerably reduced by bronchoscopic aspirations repeated twice weekly over a period of two or three months combined with continuous postural drainage, the patient lying on the abdomen at night and for the greater part of the day with the foot of the bed raised 10 in. In one case we found that the patient was dry for several hours after bronchoscopy and therefore bronchoscoped her immediately before operation, while in several cases we have slipped in a bronchoscope at the end of the operation and aspirated all secretion from the main bronchi.

We believe that bronchoscopy is an essential accompaniment in all chest surgery.

Phrenic Paralysis.—It is an advantage to have the diaphragm paralysed at the time of operation, and the paralysis should only be temporary, because if the remaining lobe is to return to its full and increased function, the diaphragm must be working. The object in paralysing the diaphragm is to reduce movement at the time of operation and so assist the surgeon when dividing adhesions. In all except three of our cases the phrenic nerve was crushed just above the diaphragm after the thorax had been opened. In *Case 6*, on account of the massive adhesions, the nerve was not seen until they had all been divided, and was therefore left intact. When there are many adhesions some time may be wasted in finding the nerve in the chest. We think therefore that in future it will be wisest to crush the nerve in the neck a few days beforehand, as we did in *Case 9*.

Artificial Pneumothorax.—In five of our cases we attempted to obtain a pneumothorax beforehand, but owing to adhesions this is often impossible and failure to obtain a space does not indicate the type of adhesion, nor is it any contra-indication to operation. The idea that a preliminary pneumothorax adjusts the patient for an open thoracotomy we find to be a theoretical rather than a practical consideration. We have therefore abandoned preliminary pneumothorax, and at the time of operation if there are no adhesions we allow the air to enter the pleural cavity slowly through a small opening.

OPERATIVE TECHNIQUE.

Anæsthesia.—There are two essential points in the anæsthesia: (1) After opening the chest, the lung on the affected side must be allowed to collapse and give ample room for manipulation; (2) Quiet respirations, as violent movement of the lung causes a flopping of the mediastinum and shock.

Shenstone and Janes advocate spinal anæsthesia. In our first two cases we employed 'avertin', 0.11 grm. per kilo. of body weight, but this has the disadvantage that the respirations are very shallow—in fact continuous oxygen must be administered while the chest is open—but it has the advantage that one does not require an expert anæsthetist.

For our last nine cases we have given a preliminary injection containing morphine gr. $\frac{1}{6}$, hyoscine gr. $\frac{1}{150}$, and atropine gr. $\frac{1}{100}$, and nitrous-oxide-oxygen during the operation at a very slight positive pressure: the percentage of oxygen varies from 18 to 25: there should be the minimum of re-breathing

as the CO₂ causes increased movement of the lung. This we think is the ideal if one has an anæsthetist with considerable experience and a McKesson's type of apparatus.

For forty-eight hours before operation the patient is given abundant glucose in all drinks and provided with barley sugar to eat. Towards the end of the operation the patient is given 1 litre of 5 per cent glucose at 115° F. per rectum.

Position of Patient.—The patient lies on the good side on a flat table, but if there is much secretion in the trachea the table is tilted to about 15° Trendelenburg position to encourage drainage from the mouth.

Incision.—Our experience so far has been confined to lobectomies for disease in the lower lobe. Although we have exposed the upper lobe on several occasions by this approach we have not yet performed an upper-lobe lobectomy. The technique, however, would be identical except that the chest is opened through the fourth interspace instead of the seventh. In the following description we are considering a lower-lobe lobectomy.

The incision through the skin and extracostal muscles runs in a straight line from the costal margin in front along the seventh interspace to the mid-line behind. All bleeding points in the skin, subcutaneous tissue, and muscle are carefully picked up with artery forceps, and we have found it a considerable saving in time to coagulate these vessels with a diathermy current rather than to tie each one with catgut. The lateral margin of the erector spinæ is then freed, and the posterior ends of the 7th and 8th ribs are exposed. Next the intercostal muscles in the middle of the incision are divided for about 3 in., the space is widened by pulling the ribs apart with small hook retractors, and so the parietal pleura is exposed and incised. If there is a rush of air into the chest, the opening is immediately plugged with the finger and the air allowed to enter slowly during an interval of a minute or two. Having found a pleural space, the intercostal muscles and pleura are then rapidly divided for their entire length with a pair of scissors. At the posterior end this incision keeps near the lower rib so as to avoid the intercostal vessels. The erector spinæ is now retracted, and with a pair of straight bone-cutting forceps the posterior ends of the ribs above and below the incision are divided opposite the tips of the transverse processes without previous elevation of the periosteum. Before the ribs are spread apart pleural adhesions in the neighbourhood must be separated. The fingers of the two hands are then inserted into the chest and the ribs slowly but forcibly pulled apart, for 4 to 5 in. in an ordinary adult. An illuminated rib retractor^{11*} is then inserted and opened out to maintain this space.

* This instrument is a modification of the ordinary abdominal retractor. The arms are longer and slightly curved so that they will lie against the chest wall; also the blades are 2½ in. wide and curved to a semicircle. In addition it carries a 3-volt lamp on each side for illumination. The light carriers, which are curved to the shape of the blades, clip on to the arms, and at their other ends are connections for the electric leads. These leads are rubber-covered and sterilized by boiling. The lights will illuminate 'round the corner' where the operating-room lights cannot reach, and this is a great assistance when dividing adhesions in the costo-phrenic sinus or apex prior to lobectomy. The lights on the retractor are not sufficient to replace all other illumination during the operation.

Pleural Adhesions.—In lobectomies performed for bronchiectasis we have encountered adhesions in every case: between base and diaphragm only in 2 cases, completely surrounding the lower lobe but not the upper in 5 cases, and with both lobes involved in 2 cases. In *Case 8*, for carcinoma, there were no adhesions.

These adhesions are usually thin and sheet-like and practically avascular, with the exception of those which run from the inferior surface of the lobe to the diaphragm, which are thicker and often contain blood-vessels that require to be ligatured. Early in the series we abandoned a case on account of the extensive adhesions, but since our experience has increased we have done cases just as bad as this; in fact, we doubt if there are any adhesions which cannot be overcome by careful dissection.

All adhesions surrounding the lower lobe must, of course, be separated, and it is advisable to free the costal surface of the upper lobe in order to allow it to expand. The lobes are separated along the oblique fissure, but it is unnecessary to split this right down to the vessels unless there is a natural plane of separation. Finally the pulmonary ligament is divided up as far as the inferior pulmonary vein, and in doing this one may encounter one or two blood-vessels that require to be ligated. A pedicle will now be produced round which the operator's finger and thumb should be able to meet. We have a special pair of lung forceps and long scissors with curved points which we find excellent for dissection and division of adhesions. Care should be taken not to draw up the diaphragm and cut it when dividing basal adhesions; when working in front of the vertebral bodies the extreme proximity of the opposite pleura in this position must be remembered, and in this region the œsophagus on the left and inferior vena cava on the right must be noted and avoided.

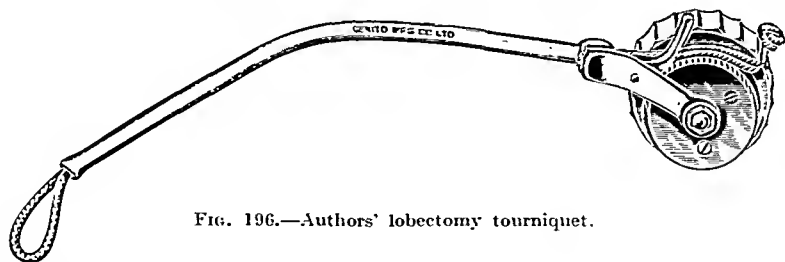


FIG. 196.—Authors' lobectomy tourniquet.

Application of Tourniquets and the Removal of the Lobe.—The loop of a tourniquet* (*Fig. 196*) is slipped over the diseased lobe and kept as high up the pedicle as possible while the assistant winds in the cord and tightens it as much

* The authors' tourniquet consists of a tube which is oval in section, is 9 in. long, and bent in the middle to an angle of 140° . The end that comes against the pedicle is slightly splayed out and has thick lips, while at the other end is a drum on which the cord of the tourniquet is wound. The tube is threaded, from the drum end, with a loop of thick blind cord, the loose ends of which are knotted together and are then attached to the drum by fitting into a groove on the rim. The drum will wind in either direction, but when the ratchet-catch is applied the cord can only be wound up. In practice the catch is not used until most of the loop has been wound in; it is then applied and the cord steadily tightened up round the pedicle.

as he can. A second tourniquet is then employed and tightened up round the lung $1\frac{1}{2}$ in. distal to the first (*Fig. 197*). The pleural cavity is packed off with four or five large dry gauze swabs, which also cover over the margins of the wound in the chest wall. The pedicle is then divided half-way between the cords with a pair of curved seissors. Whilst dividing this the surgeon must constantly note his relation to the proximal tourniquet, and the assistant will find that he can tighten up the cord two or three more points

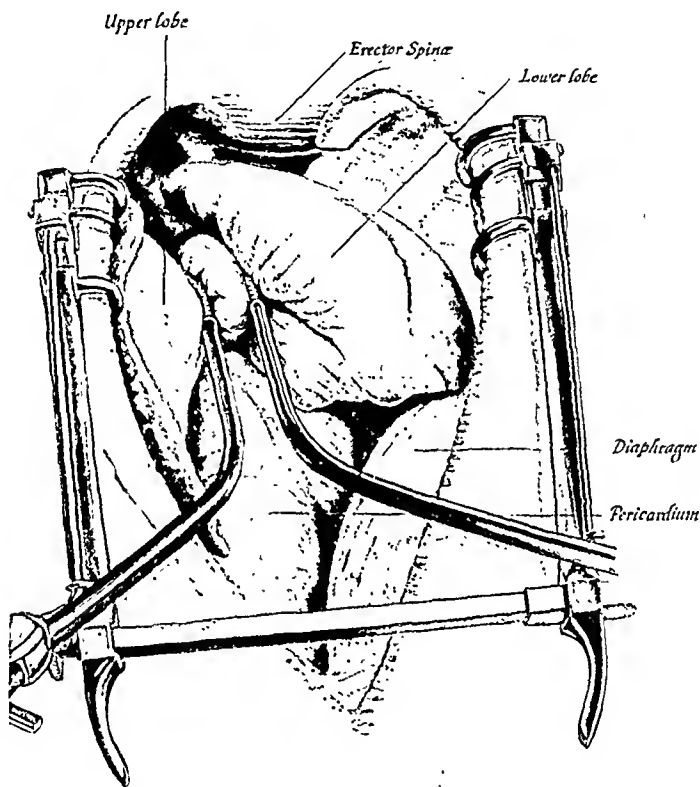


FIG. 197.—The two tourniquets in position.

on the ratchet. Any pus that escapes must be carefully mopped up with small swabs on holders, and when the lobe has been removed the pedicle is swabbed with 1-1000 flavine and then the gauze packs are removed.

Closure of the Pedicle.—The cut end of the pedicle projects for about $\frac{1}{2}$ in. beyond the proximal tourniquet and presents a central white area consisting of blood-vessels and bronchi and a small margin of grey lung tissue (*Fig. 198*). The pedicle is sutured with No. 1 chromicized catgut on a round-bodied half-circle needle. This passes through the white broncho-vascular area as if this was a homogeneous structure and draws it together; the fringe of lung is not included in the first two rows (*Fig. 199*). The suture is continuous; it starts at one end of the pedicle, and after reaching the other

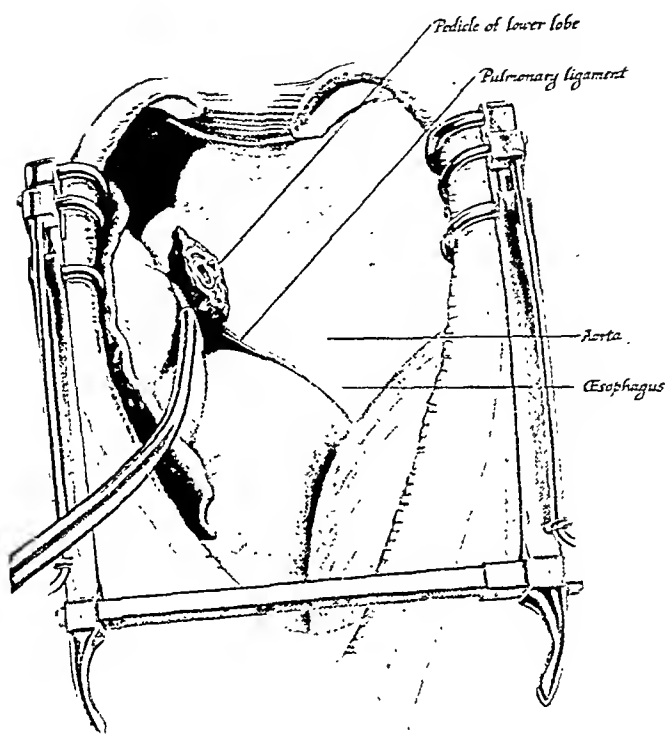


FIG. 198.—The pedicle after removal of the lower lobe.

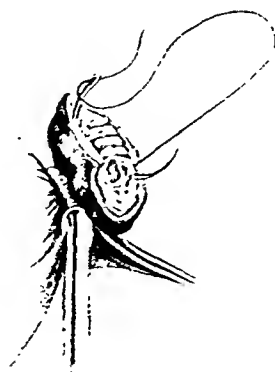


FIG. 199.—The first row of the continuous suture closing the pedicle (semi-diagrammatic).

end returns as a second row to the first point, where it is tied. The vessels and bronchi are thus closed, and the tourniquet is now relaxed a little and if there is no hæmorrhage is completely removed. There may, however, be one or two points on the pedicle which bleed and require to be picked up with forceps and sutured, but usually no bleeding of consequence occurs. A third continuous suture then draws the fringe of lung tissue across the surface of the pedicle, and when this is complete the pedicle is buried into the under surface of the upper lobe by three or four sutures.

Before removing the retractor the pleural cavity must be carefully inspected for any oozing vessels, the most frequent sites being on the diaphragm, along the pulmonary ligament, or posteriorly where the ribs have been divided. Here the diathermy is of the utmost assistance in coagulating areas which are difficult to tie.

Drainage.—The rib retractor is next removed. The skin and extracostal muscles are retracted, the lateral surface of the 9th rib is exposed in the mid-axillary line, and an inch of the rib excised by the ordinary subperiosteal resection. A stab incision is made through the skin and extracostal muscles into the chest and a drainage tube is drawn from within outwards. The intrathoracic portion of this tube has several side openings and is about 4 in. long and is attached to the diaphragm by a loop of catgut so that the tip lies an inch below the pedicle. Shennstone and Janes advocate an ordinary stab incision without resection of a piece of rib, but we have found that if the tube is large enough for free drainage it causes pain from pressure on the intercostal nerve and may become obstructed.

Closure of Chest.—Pericostal sutures are inserted $1\frac{1}{2}$ in. apart for the whole length of the wound; we prefer to pass these through the centre of



FIG. 200.—The rib approximator in position and two pericostal sutures.

the interspace for fear of puncturing the intercostal vessels. The most posterior of these sutures passes through the lateral margin of the erector spinæ, then round the two ribs and through the erector spinæ again. The ribs are then drawn together by the rib approximator* (*Fig. 200*), the

* The authors' rib approximator consists of two hook retractors which are united together by a bolt that runs in a groove on the shaft of the two retractors: they can be quickly locked or released by a half turn of the bolt.

ligatures tied, and the instrument is removed. The extracostal muscles are sutured with one long continuous suture taking all layers together, and the skin by another continuous suture.

TIME AND SHOCK.

We have found that neither the blood-pressure falls nor the pulse-rate rises when the chest is opened, when the tourniquet is applied, or when the pedicle is divided. The amount of shock is proportional to the extent of the adhesions, because on them depends the length of the operation and the amount of bleeding. For example, there were no adhesions in *Case 8*, and although the man was 55 he left the table with a pulse of 132 and systolic blood-pressure of 120, whereas in *Cases 6 and 7* there were very extensive adhesions so that the operation lasted nearly two hours in each case, and there was considerable shock at the end.

We agree with Lilienthal that in a major thoracotomy more than in any other operation in surgery time is of the utmost importance. He states that about forty-five minutes is the maximum, but from our experience we find that we can safely take twice as long before the blood-pressure begins to fall and the pulse-rate to increase. The accompanying chart (*Fig. 201*) of blood-pressure and pulse-rate shows a phenomenon that we have observed in most of the cases where these records have been kept, i.e., a rise of blood-pressure during the first hour of operation; and it is not until after an hour to an hour and a half that the blood-pressure begins to fall and the pulse-rate to increase. Towards the end of the operation there is always an improvement when the chest has been closed.

We find that our average time of operation is one and a half hours, the shortest time being one hour and the longest just over two hours. Time may be saved during the course of the operation by the use of a diathermy current for coagulation of blood-vessels, the help of an experienced assistant who can divide from his side the adhesions which the surgeon cannot reach, and the closure of the extracostal muscles and skin by continuous sutures.

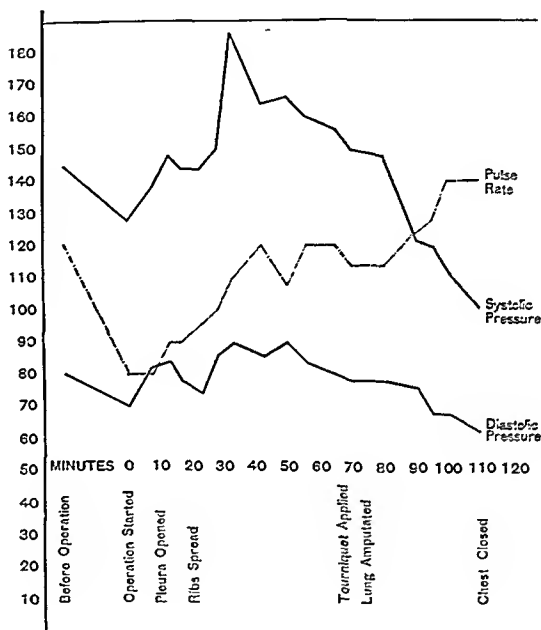


FIG. 201.—*Case 7*. Chart of blood-pressure and pulse-rate during operation.

Other factors in the prevention of shock are a rigid attention to hæmostasis and a quiet anæsthesia with avoidance of excessive mediastinal movement.

POST-OPERATIVE TREATMENT.

Blood Transfusion.—It is advisable to have the patient grouped before operation and a blood donor available at short notice, and in those cases where the operation has lasted more than one hour we have usually given a transfusion of 300 to 400 c.c.

Position in Bed.—The patient is put in the sitting-up position so that any pleural effusion will gravitate to the bottom of the cavity and be removed by the drainage tube.

The Drainage Tube.—We use a Winchester bottle with a wide neck and rubber cork through which passes one short glass tube and one long one extending down 2 in. under the water in the bottle. This bottle and the water should be sterile, and it is placed on the floor beside the patient's bed. As soon as the patient returns to the ward the drainage tube is connected with the glass tube going under the water, and the air bubbles out as the lung expands. The rise and fall of the fluid in the glass tube indicates that the drainage tube is patent, and if this becomes obstructed it must be manipulated until it works. Care must be taken when the drainage tube is first attached to the bottle, especially when the diaphragm has not been paralysed, as there may be a high negative pressure within the chest. In one such case the fluid in the bottle was seen to be sucked back, and unless the tube had been rapidly clipped would have entered the chest. A screw clamp, which narrows down the tube considerably will obviate this, while still allowing respiratory movement and drainage. On no account must air be allowed to escape into the chest during the first ten days, after which time the upper lobe will be adherent to the chest wall and the water bottle may then be disconnected. It is imperative that the drainage tube be kept patent during the first fourteen days to prevent the danger of a positive pressure pneumothorax which might result if the bronchial stump opened and there was no escape for the air. We do not, however, know of any cases where this complication has occurred in any operations performed by this technique.

POST-OPERATIVE COMPLICATIONS.

Primary Hæmorrhage.—This has occurred in two of our cases. In *Case 5* the chest had to be opened up the same evening and a small spurting artery was found in the pulmonary ligament and ligatured. As no vessel had been ligatured in this position during operation it must have been temporarily occluded, as the chest was dry before being closed. In *Case 7* the bleeding was a slight ooze and was controlled by the injection of 20 c.c. of 3 per cent 'coagulin' and the clamping of the drainage tube for twelve hours.

Secondary Hæmorrhage.—This occurred only in *Case 10*. On the fourteenth day after operation a sudden and severe bleeding took place, three pints of blood collecting in the bottle. The tube was clamped and the man given a blood transfusion. On opening the chest a basal empyema cavity

was found which contained blood-clot, but the bleeding had stopped. In view of the rapidity of onset it is probable that the blood had come from the pedicle: it is noteworthy that this is the only case in which the pedicle was not buried in the under surface of the upper lobe at the end of the operation.

Empyema.—Three cases have developed a basal empyema. In *Cases* 6 and 7 this was quite small, but in *Case* 10 the cavity was about 4 in. in diameter. No patient, however, was severely ill from the infection. The empyema has rapidly healed in each case, with the exception of the last one, which is too recent to report finally.

Infection of the Chest Wall.—Infection occurred in three cases. In *Cases* 5 and 10 it followed re-opening of the chest wall for hæmorrhage. *Case* 6 developed a localized abscess in the chest wall which had to be incised, and a sinus was then found leading into the pleural cavity at the posterior end of the wound; after drainage the pleural sinus closed within a week.

Subphrenic Abscess.—Subphrenic abscess occurred in *Case* 10, and was due to injury to the diaphragm at the time of operation.

Bronchopneumonia.—This developed in one patient only (*Case* 5), who had had a severe primary hæmorrhage.

Bronchopleural Fistula.—An obvious blowing fistula has occurred in three cases only (*Cases* 6, 7, and 10). Since our third case we have injected lipiodol into all the sinuses, and in two cases (*Cases* 4 and 8) we discovered a small bronchial fistula which had been entirely unsuspected. As the fistulous track is a long one, it tends to heal rapidly as the drainage tube is gradually shortened.

Cerebral Abscess.—In *Case* 1 the patient died from a suspected frontal abscess ten days after operation, and at autopsy an abscess was found in the left frontal lobe. It was 2 in. in diameter and well localized with thick walls, and on this account the pathologist considered that it must have been present before operation.

REPORTS OF CASES.

Case 1.—Sarah W., aged 13, schoolgirl, admitted to St. Bartholomew's Hospital on May 9, 1932 (referred by Dr. R. Hilton). Her father had died three years previously from pneumonia. There had been nineteen children in the family, and thirteen of these had died from pneumonia.

HISTORY.—At the age of 2 the child had diphtheria, for which tracheotomy was necessary, and this was followed by pneumonia; ever since then she had had a chronic cough. At 5 and at 11 years of age there were further attacks of pneumonia. For the past fifteen months the sputum had been becoming more copious, for the past nine months it had been foul, and for five months there had been shortness of breath. Apart from this, the child's general health had been good and her weight had steadily increased.

ON EXAMINATION.—A red-faced plump child, marked fætor of the breath, weight 74 lb., no pyrexia, pulse 90, respirations 25. There was an old interstitial keratitis of the right cornea. The heart was displaced to the left, but the sounds were natural. The chest showed poor expansion, most marked at the left base. There was impairment of percussion note at both bases, and diminished breath-sounds. At the left base there was an area of bronchial breathing; there were

many râles, but more on the left than right. In the abdomen neither the spleen nor the liver was palpable. The urine contained no albumin. An X-ray showed that the heart was displaced to the left, and the left diaphragm was slightly higher than normal. The curved outer margin of an atelectatic left lower lobe could be seen through the heart's shadow. Lipiodol demonstrated a severe bronchiectasis in the collapsed lobe, while the rest of the two lungs was healthy (Fig. 202).



FIG. 202.—Case 1. Lipiodol demonstrating congenital bronchiectasis of the left lower lobe.

OPERATION (May 25, 1932).—Left lower-lobe lobectomy. Anaesthetic (by Mr. Rait-Smith), avertin and continuous oxygen after the chest was opened. Incision through the seventh interspace on the left side. The phrenic nerve was crushed where it lay on the pericardium. The left lower lobe presented a marked contrast to the upper lobe: it was diminutive in size and of a dark purplish-grey colour. There were only a few adhesions over the costal surface of this lobe, but firm adhesions to the diaphragm and in the interlobar fissure. These were separated without much difficulty and the lobectomy performed. The chest was drained by the insertion of a tube through a stab incision in the ninth interspace in the posterior axillary line. The operation lasted about one and a half hours.

POST-OPERATIVE COURSE.—The patient had practically no post-operative fever; the pulse-rate varied from 52 to 110; the respirations were at an average of 30 per minute. The child complained of a considerable amount of pain on the left side, which we think was due to the inter-

costal drainage tube. The general condition was, however, very satisfactory, but on the fourth day after operation the child appeared to be listless and looked as if her head was too heavy. The next day she had severe left frontal headache and was drowsy. There was definite drooping of the left eyelid, but on examination of the nervous system the only abnormal sign to be discovered was an extensor plantar response of the right foot, although this sign did not appear to be constant. On this day the pulse-rate was 110, and the blood-pressure 135/75. The headache persisted and the patient was drowsy at one time and restless at another. No further physical signs developed. A cerebral abscess was suspected, but in the absence of more definite signs craniotomy was considered inadvisable. On the night of the ninth day the child was restless, and then became listless and drowsy, and did not notice the things about her. The right pupil was very dilated and the left eyelid was drooping. Early the next morning the child suddenly started to choke, and pus appeared from the nose and was also coughed up from the mouth, and she died within a few minutes.

POST-MORTEM EXAMINATION.—This revealed a large abscess in the upper part of the left frontal region of the brain, reaching right through to the cortex, with marked flattening of the convolutions. This abscess had a fibrous wall about 3 mm. thick, and from its appearance the pathologist thought it was probably of more than ten days' duration. The examination of the thorax showed that the right pleural cavity was obliterated by adhesions, the underlying lung was healthy and there was no bronchiectasis present. On the left side the lower lobe had been removed. In the region of the pedicle there was a small potential space, but no fluid or sepsis was present. The upper lobe was expanded to fill the rest of the

hemithorax, and its lower surface was adherent to the diaphragm. The lung tissue itself appeared to be quite healthy.

PATHOLOGICAL REPORT.—On section the diseased left lower lobe presented the typical appearance of a congenital atelelectatic lobe—namely, a number of thick-walled dilated bronchi surrounded by fibrous tissue, with almost complete absence of any parenchyma (*Fig. 203*).

Case 2.—Mary R., aged 23, clerk, admitted to the Brompton Hospital on April 22, 1932 (referred by Dr. Wall).

HISTORY.—Quite well until 1930, when she had a sudden hæmoptysis. Since then she has continued to spit blood in clots at least once a week, and has noticed that it is worse a day or two before menstruation. Coughs only when bleeding. Has lost weight recently. Admitted to the Brompton Hospital in July, 1931, under Dr. Wall.

ON EXAMINATION.—There were no physical signs in her chest; her heart was normal; blood-pressure 120/90. X-ray on July 31 showed that the heart and mediastinum were displaced to the left but no infiltration of the lungs. After the injection of lipiodol and a more heavily exposed film a dense opaque area could be seen through the cardiac shadow and the lipiodol failed to enter the left lower bronchus.

The patient was therefore referred to one of us (J. E. H. R.) for bronchoscopy. This demonstrated that the left lower-lobe bronchus was completely blocked by a very vascular growth, which bled profusely when a piece was removed for section. The section of the biopsy specimen showed chronic inflammatory tissue only. Further bronchoscopies performed a month later demonstrated a stenosis at the entrance to the left lower lobe. The patient was therefore bronchoscoped on seven further occasions and the stricture was dilated with bougies and granulation tissue painted with silver nitrate. In spite of this treatment the patient still continued to spit up blood. Therefore exploratory thoracotomy was advised with a view to lobectomy. On May 17 an artificial pneumothorax was induced on the left side.

OPERATION (May 26, 1932).—Left lower-lobe lobectomy. Anæsthetic (by Mr. Hewer), avertin and nitrous-oxide-oxygen. Incision through the seventh intercostal space; the phrenic nerve was crushed just above the diaphragm; the left lower lobe was small and airless and of a firm consistency; there were a moderate amount of adhesions surrounding this lobe; it was impossible to use the distal tourniquet owing to the consistency of the lobe. At the end of the operation the chest was drained by a stab incision in the ninth interspace.

POST-OPERATIVE COURSE.—This was uneventful except for some intercostal pain owing to the drainage tube. This was removed on June 6. The patient left hospital completely healed on July 10. The patient was seen in the Out-patient Department on April 28, 1933. She stated that during the last three months she had had four hæmoptyses. They all occurred at night when she was in bed, and she attributed them to nose-bleeding. Also since leaving hospital she had lost 6 lb. in weight. She had no cough or sputum, and no pain except slightly during damp weather. On examination she looked thin but healthy; there was a good air entry at her right base. Under the X-ray the diaphragm was high, the movements were normal but limited in extent; the heart was displaced to the left, and there was some pleural thickening in the left lower zone, but apart from this no abnormal shadow could be seen.



FIG. 203.—*Case 1.* Section across left lower lobe, showing a congenital type of bronchiectasis.

PATHOLOGICAL REPORT.—On section the diseased lobe showed that the main lower-lobe bronchus was dilated to about an inch in diameter and projecting into it was a bright yellow necrotic looking growth. The rest of the lung tissue was collapsed. The microscopic section showed that the growth was undoubtedly malignant and probably a squamous-celled carcinoma of undifferentiated type.

Case 3.—Annie R., aged 43, housewife, admitted to the Brompton Hospital on April 26, 1932 (referred by Dr. Bosanquet).

HISTORY.—Pneumonia in childhood. Eight years previously was under observation in hospital for pulmonary tuberculosis. Has had a cough off and on for years. Three years previously had pleurisy, but does not remember which side. In April, 1932, the cough started again and she had pain in the left side of her chest and several small hæmoptyses. On admission she had 2 oz. of purulent sputum a day, with dyspnoea on exertion and pain in the left side of her chest.

ON EXAMINATION.—A thin but healthy-looking woman, weight 115 lb. Heart slightly displaced to the left, but sounds normal. The lungs gave an impaired note at the left base, with bronchial breathing and many coarse râles. Abdomen normal. Sputum was negative for tubercle bacilli. X rays showed the heart displaced to the left, fibrosis of the left base, and a suggestion of bronchial dilatation in the left lower lobe. This was confirmed by lipiodol (Fig. 204). There was no evidence of dilatation in any other lobes of the lung. The patient was under observation in hospital for three months, during which time she gained weight and the sputum decreased to 2 drachms daily. An artificial pneumothorax was induced on July 18 and refilled every other day.



FIG. 204.—Case 3. Lipiodol demonstrating acquired bronchiectasis of the left lower lobe.

fissure, which made the operation extremely simple. The chest was drained through a stab incision in the eighth interspace in the mid-axillary line.

POST-OPERATIVE COURSE.—This was entirely uneventful. The tube was removed on the fourteenth day, and from this day the patient began to get up. She left hospital completely healed on the twenty-eighth day after operation. In December, 1932, she was seen and was well with no cough or sputum. On May 28, 1933, the patient stated that her general health was excellent, and this winter was the first she had gone through without having a cold. She had no cough, but said she occasionally brought up sputum in the morning; no hæmoptyses, but occasional pain in the chest during damp weather. On examination the chest was symmetrical and moved equally on the two sides; a good air entry was noted and no added sounds. Under the X-ray the left diaphragm was seen to be raised and fair movement was present on inspiration. The heart was displaced to the left, and there was some fibrosis at the left base.

PATHOLOGICAL REPORT.—On section the diseased lobe presented the typical appearance of an acquired bronchiectasis in the lower and medial part of the lobe

OPERATION (July 28, 1932, at the Centenary Meeting of the B.M.A.).—Left lower-lobe lobectomy. Anæsthetic (by Mr. Hewer), nitrous-oxide-oxygen. Incision through the sixth interspace. The phrenic nerve was crushed just above the diaphragm. The lower lobe was of normal size and appearance when compared with the upper lobe. There were no adhesions except for a few between the lower lobe and the diaphragm and in the interlobar

—namely, very little peribronchial fibrosis, six to eight dilated bronchi with thick walls, while the rest of the lung showed normal healthy-looking parenchymal tissue. An illustration of this type is shown in *Fig. 209*.

Case 4.—Charlotte S., aged 32, housewife, admitted to the Brompton Hospital on July 25, 1932 (referred by Dr. Burrell).

HISTORY.—Left-sided pneumonia in November, 1931, followed by cough and purulent offensive sputum, which has persisted. In December, 1931, there was an hæmoptysis of one pint. On admission she still had the cough and a trace of sputum which was slightly blood-stained. There were also occasional pains in the left side of the chest.

ON EXAMINATION.—A well-grown thin woman of a good colour, weight 111 lb. No clubbing of fingers. Heart normal; lungs showed poor movement and impaired percussion note at the left base behind, with bronchial breathing over this area, increased voice conduction, whispering pectoriloquy, and a few coarse râles. X rays



FIG. 205.—*Case 4.* Lipiodol demonstrating advanced bronchiectasis of the left lower lobe.



FIG. 206.—*Case 4.* Lateral view.

showed the heart slightly displaced to the left, fibrosis at the left base with suggestion of bronchial dilatation. This was confirmed by lipiodol, which showed a very marked basal bronchiectasis involving the left lower lobe only (*Figs. 205, 206*). Pre-operative artificial pneumothorax was attempted but not obtained.

OPERATION (Aug. 27, 1932).—Left lower-lobe lobectomy. Anæsthetic (by Mr. John Hunter), nitrous-oxide-oxygen. Chest opened through the sixth interspace. There were many thin adhesions over the lower lobe and a few over the upper. These were all divided. The phrenic nerve was then found and crushed just above the diaphragm. The lower lobe was firm and airless, smaller and of a darker colour than the upper lobe. At the end of the operation the chest was drained through a stab incision in the seventh interspace in the mid-axillary line. The operation lasted about one and a half hours and the condition at the end was very good.

POST-OPERATIVE COURSE.—On Sept. 17 the patient had been running a temperature of 100° every evening since the operation. Therefore an X-ray was taken on this day, which showed a small fluid level at the base. The tube was

therefore removed and replaced by a catheter. Following this the temperature returned to normal. On Sept. 27 lipiodol was injected into the sinus and demonstrated a long track running upwards towards the root of the lung and a small unsuspected bronchial fistula. The patient was discharged two days later with a small tube. The tube was removed a week later and the sinus promptly healed. On Jan. 6, 1933, the patient was seen again. She was very well and had no symptoms. There was a poor air entry at the left base. X rays showed nothing abnormal except a rather high diaphragm with very limited but normal movement (*Fig. 207*). On April 28, 1933, the patient had no symptoms referable to her chest; her general health was good and she was gaining weight; no added sounds in the lung. Under the X-ray screen the left diaphragm was of normal level; the movements on the two sides were practically equal, but the left diaphragm was held up slightly in the costo-phrenic angle. Otherwise nothing abnormal.



FIG. 207.—*Case 4.* X-ray of the chest four and a half months after left lower-lobe lobectomy.

remaining pulmonary parenchyma (*Fig. 208*).

Case 5.—Ernest W., aged 19, clerk, admitted to St. Bartholomew's Hospital on Oct. 8, 1932 (referred by Dr. Geoffrey Bourne).

HISTORY.—Twelve years previously he had had pneumonia; then quite well until five years ago, when he developed a cough with a little sputum; this did not bother him, and his general health remained good until one year ago, when he coughed up about half a cupful of blood. Since then has had a nervous breakdown, the cough became worse, the sputum increased, and he lost weight.

ON EXAMINATION.—A tall youth, thin but healthy-looking, weight 142 lb. Slight clubbing of nails. The chest moved equally, with a slight flattening on the left side; left base percussion note impaired, air entry less than the right; harsh breath-sounds with a few added sounds. X rays without lipiodol showed nothing abnormal,

PATHOLOGICAL REPORT.—On section the diseased lobe presented a marked degree of bronchiectasis throughout the whole lobe, with pneumonitis in the



FIG. 208.—*Case 4.* Section of the left lower lobe showing a marked degree of bronchiectasis with pneumonitis.

but after introduction of the oil an area of bronchiectasis was seen in the left lower lobe of moderate extent. The right side and left upper lobe were normal. A pre-operative artificial pneumothorax was induced on Oct. 18 and a moderate collapse obtained.

OPERATION (Oct. 26, 1932).—Left lower-lobe lobectomy. Anæsthetic (by Mr. Frankis Evans), nitrous-oxide-oxygen. Incision through the seventh interspace. The phrenic nerve was crushed just above the diaphragm. The left lower lobe did not present any contrast in size or appearance from the upper. There were some adhesions surrounding the lower lobe, while the upper lobe was free. The operation was performed by the usual technique, but instead of draining the chest through a stab incision in this case, we resected 1 in. of the 10th rib in the posterior axillary line, as described in the text. The operation lasted sixty-five minutes and at the end the pulse-rate was 90 and the blood-pressure 98/52.

POST-OPERATIVE COURSE.—Four hours after operation we were notified that the pulse-rate had been steadily increasing and was now 140 and there was a little over a pint of blood in the drainage bottle. It was obvious that the patient was bleeding, and it was decided to re-open the chest. A transfusion of 800 c.c. of blood was given and the patient anæsthetized with gas and oxygen. The whole incision was re-opened and a small spurting artery could then be seen at the lower end of the pulmonary ligament. It was clamped and tied. No vessel in this position had been tied during the operation so it must have been temporarily occluded. The patient developed a post-operative bronchial pneumonia at the right base and coughed up about 6 oz. of greenish sputum daily for the first two weeks, after which time he gradually got better. He also had considerable pain in the left side of the chest, and on the right side from pleurisy. He was out of bed thirty-five days after operation and left hospital on Jan. 4, 1933, at which time he had no cough or sputum, the temperature, pulse, and respirations were normal, and he said that the 'tickling sensation' in his chest had completely gone.

On May 28, 1933, the patient had no symptoms whatsoever, was gaining weight, and feeling well. He said, "It is a real boon to me to be able to go about in company and no longer feel embarrassed, as I used to before." Under the X-ray the left diaphragm was seen to be raised, but slight normal movement was present; the heart was displaced to the left, but apart from this nothing abnormal was seen.

PATHOLOGICAL REPORT.—On section the diseased lobe presented the typical appearance of an acquired bronchiectasis, and, as shown in *Fig. 209*, the upper half, i.e., that portion supplied by the dorsal bronchus, was absolutely normal, while the lower half contained the dilated bronchi and some degree of pneumonitis in the parenchyma.



FIG. 209.—Case 5. Section of the left lower lobe showing an acquired type of bronchiectasis.

Case 6.—Henry O., aged 39, warehouseman, admitted to the Brompton Hospital on Oct. 10, 1932 (referred by Dr. Thomas Nelson).

HISTORY.—Twenty-nine years previously he had had pleurisy, but does not remember which side. In August, 1931, he had twenty-three teeth extracted under a general anæsthetic, and shortly after that developed a cough, which has continued. In May, 1932, he started to have an offensive sputum, which was most noticeable in the morning or on exertion. In June had to stop work owing to pain in the right side of the chest, increase of the cough and sputum, increasing shortness of breath, and loss of weight.

ON EXAMINATION.—A thin, ill-looking man, weighing only 91 lb. He had no pyrexia. The heart was displaced towards the right; on the right side there was poor movement, impaired percussion note, weak breath-sounds, bronchial breathing, and râles both in front and behind. The rest of the physical examination was normal, and the urine contained no albumin. X rays showed movement of the right diaphragm restricted, mediastinal structures displaced to the right, left lung normal, while the right lung showed partial collapse of the lower lobe, with fibrosis and suggestion of bronchiectasis. This observation was confirmed by lipiodol, which also demonstrated some dilatation in the terminal branches of the right middle lobe. On Oct. 25 a pre-operative artificial pneumothorax was attempted, but only a small pocket could be obtained.

OPERATION (Oct. 29, 1932).—Right lower-lobe and partial middle-lobe lobectomy. Anæsthetic (Mr. Hewer), nitrous-oxide-oxygen. Incision through the seventh right interspace. Very dense and widespread adhesions throughout the hemithorax, but most marked over the middle and lower lobes. The phrenic nerve could not be found. The right lower lobe was first removed and the pedicle completely sutured. The lower margin of the middle lobe presented many hard nodular areas, but as there was no fissure between the upper and middle lobes, a middle-lobe lobectomy by the usual technique was impossible, and it was therefore decided to do a partial pneumectomy of the middle lobe. Two intestinal clamps were placed across the middle lobe and the distal two-thirds excised. There was considerable bleeding at the hilum region where the ends of the two clamps met, and artery forceps had to be applied. The cut surfaces of the lung were oversewn and the hilum closed by a continuous catgut suture. (This partial amputation of the middle lobe was very unsatisfactory and we decided never to employ it again. We consider it would have been better to have created a fissure between the upper and middle lobes by cutting into the lung with a diathermy knife and then using the tourniquets as in a lower-lobe lobectomy.) The chest was closed and drained by resection of a portion of the 9th rib. The operation lasted just over two hours, and by the time the chest was closed the patient's pulse was only just palpable at the wrist. He was therefore given a blood transfusion and intravenous saline before leaving the operating table.

POST-OPERATIVE COURSE.—The patient ran a temperature of 101.5° and developed a discharge from the upper end of his incision, and at the end of ten days there was a purulent discharge from the drainage tube. On Nov. 26 it was found that the discharging sinus at the upper end of the wound communicated with a subcutaneous abscess and also went down to the cut ends of the two ribs, which could be felt to be necrosed. This portion of the wound was laid open and the subcutaneous abscess drained dependently. Following this the patient's temperature returned to normal. On Dec 16 he had a small quantity of purulent discharge from the chest, there was an obvious bronchial fistula, and examination with a probe (confirmed by X rays) showed that there was no cavity but a small track inside the chest which led up to the fistulous opening: there was a small sinus at the upper end of the wound going down to the necrosed rib. On Jan. 10, 1933, the patient was up; the sinus at the upper end of the wound had healed; there was still a tube in the chest wall and a bronchial fistula, which, however, sounded much smaller.

On Jan. 26 another patient in the ward developed influenza, and our patient caught it. His temperature suddenly rose and he had fine crepitations in the left lower lobe. On Feb. 2 he died with influenzal bronchopneumonia.

POST-MORTEM EXAMINATION.—This showed that the right side of the chest was occupied by the upper lobe and the pleura was universally adherent. The remaining lobe showed old healed tubercle at the apex, some œdema but no pneumonia. On the left side the upper lobe showed healed tubercle at the apex, but otherwise normal. The left lower lobe showed bronchopneumonia in the stage of red hepatization.

PATHOLOGICAL REPORT.—On section the diseased lobes presented an appearance very similar in type to that found in *Case 4*—namely, a severe degree of bronchiectasis with much pneumonitis in the surrounding lung tissue.

Case 7.—Robert M., aged 24, optical worker, admitted to the Brompton Hospital on Oct. 26, 1932.

HISTORY.—Eight years previously he had suffered from sudden hæmoptysis which lasted for two weeks; following this he was quite well and returned to work. Had occasional cough and slight sputum, but not sufficient to bother him. Five years ago he had another hæmoptysis similar to the first. In September, 1932, he had his third hæmoptysis, which cleared up after a few days, and he felt quite well; no cough, pain, or shortness of breath.

ON EXAMINATION.—A healthy-looking, well-covered man: heart normal, blood-pressure 160/76. The lungs showed slight impairment of percussion note at the left base, with bronchial breathing and bronchophony, but no added sounds. The white blood-count was 8700. The X rays showed that the heart and mediastinum were displaced considerably to the left and that the left base was opaque so that the diaphragm could not be distinguished. On introduction of lipiodol large pools of the oil collected at the base, which suggested that the oil was in the stomach, but it was eventually decided that it was in the lung (*Fig. 210*).

OPERATION (Nov. 5, 1932). — Left lower-lobe lobectomy. Anæsthetic (Mr. John Hunter), nitrous - oxide - oxygen. Incision through the eighth interspace. There were a very large number of short strong adhesions throughout the hemithorax. The lower lobe was dissected out with extreme difficulty. The phrenic nerve was found when sufficient adhesions had been divided and was crushed just above the diaphragm. There was troublesome bleeding from the loose cellular tissue of the mediastinum in the region of the œsophagus which was eventually stopped by hot packs. At the end of the operation a drainage tube was inserted by the resection of one inch of the 9th rib in the mid-axillary line. The operation lasted two hours, but the blood-pressure did not fall nor the pulse-rate rise until the last half hour. At the end of the operation the pulse was 140 and the systolic pressure below 80.

POST-OPERATIVE COURSE.—The patient was returned to the ward and given an immediate transfusion, following which the pulse-rate fell to 120 and the blood-pressure rose to 85. Six hours later the pulse-rate had risen to 124 and there was a definite trickle of blood coming from the drainage tube in the chest: 20 c.c. of 3 per cent 'coagulin' were introduced through the drainage tube, which was then clamped. Before this was done it was noted that the breath-sounds were strongly audible over the left upper zone, which suggested that the lung had already expanded. During the next ten days there was a blood-stained discharge from the chest. On



FIG. 210.—*Case 7.* Lipiodol demonstrating large cystic cavities in the left lower lobe. Patient standing.

Dec. 1, as the patient had been running an evening temperature of 100° to 101° , an X-ray was taken which showed that the drainage tube was kinked and there was a small collection of fluid. The tube was adjusted and the temperature fell to normal. On Dec. 9 he had an obvious bronchial fistula, with air bubbling out of the drainage tube on coughing. The tube was therefore disconnected from the bottle and he started to get up. Lipiodol on Dec. 14 showed that there was a small basal empyema with fistula. On Jan. 10, 1933, there was a small sinus in the chest wall and a fistula: practically no discharge; the patient was up and well; no hæmoptysis.

When seen on April 28 the patient stated that his fistula had healed three weeks previously. He had a sinus in his chest wall about 2 in. long; the small drainage tube,



FIG. 211.—Case 7. Section of the left lower lobe showing a congenital cystic type of bronchiectasis.

therefore, was removed. He had no symptoms. Under the X-ray the diaphragm was seen to be raised, and had a slight paradoxical movement. The heart was displaced to the left, but otherwise there was nothing abnormal. On May 5 the sinus was completely healed.

PATHOLOGICAL REPORT.—On section the diseased lobe contained two very large cysts and several smaller ones (*Fig. 211*). Microscopic section showed that the epithelium was similar to that found in the trachea, with long cilia.

Case 8.—George K., aged 55, bus driver, admitted to the Brompton Hospital on Sept. 14, 1932 (referred by Dr. R. A. Young).

HISTORY.—For many years he had had slight cough with expectoration in the morning. Three years previously, i.e., in 1929, he first spat up a little blood, and since then this had occurred on many occasions. In May, 1932, he was treated for hæmorrhoids by injection. Since then he had noticed that he had been losing weight amounting to 28 lb., and had had general weakness and malaise. On admission he had a dry cough, no sputum, but pain in the right chest.

ON EXAMINATION.—A large, rather thin-looking man and in no distress; weight 164 lb.; no clubbing. His chest was emphysematous; heart not displaced; arteries somewhat thickened and tortuous; blood-pressure 130/80. On the right side in the lower part of the axilla there was an area of slightly impaired percussion note, with weak breath-sounds and fine râles; otherwise the chest was normal. Apart from this, nothing abnormal was discovered on general examination, and the urine was normal. Sputum was negative for tubercle. Blood-count normal. X-ray

examination showed mediastinum and diaphragm normal: in the lower zone on the right side there was an irregular rounded opacity which extended out to the chest wall but was not continuous with the mediastinum (*Fig. 212*). In a lateral view it was shown to be just posterior to the heart shadow and with a peaking of the diaphragm opposite it. Lipiodol failed to enter this area. Bronchoscopy on Oct. 15 showed no evidence of enlargement of the mediastinal lymphatic glands and nothing abnormal was discovered. While under observation in hospital the patient started to have purulent sputum up to 1 oz. daily. He had a temperature of between 99° and 100° on most evenings and a leucocytosis of 15,700. The diagnosis rested between a chronic lung abscess or growth, but on account of these findings it was considered that the former was the more probable.

OPERATION (Nov. 5, 1932).—Operation for first stage of lung abscess under local anaesthesia. Five inches of the 7th and 8th ribs were resected in the mid-axillary line, and when resecting the intercostal bundle between these two the pleura was accidentally torn. The lung was then seen to be completely free and the pleural opening was therefore enlarged and the lower lobe palpated. On the outer surface of this lobe was a white scarred area about 1½ in. in diameter, and on palpation a hard spherical lump about 3 in. in diameter could be felt in the lower lobe. The diagnosis between chronic abscess and carcinoma was still in doubt, but it was decided that lobectomy could be very rapidly performed and would be the wisest course. Therefore the patient was given nitrous-oxide-oxygen anaesthesia by Mr. Seadding (the Assistant Medical Officer), the incision was enlarged forwards and backwards, and a *right lower-lobe lobectomy* performed. In cutting across the pedicle no enlarged lymphatic glands were discovered. During the first half of the operation the patient's blood-pressure steadily rose, the operation was completed in one hour, and when the patient left the table the systolic blood-pressure was 120 and the pulse only 132.



FIG. 212.—*Case 8.* X-ray of the chest showing an irregular rounded opacity in the lower zone of the right lung.

POST-OPERATIVE COURSE.—In spite of this man's age he had a more satisfactory post-operative course than any of the other cases. Six hours after operation his pulse-rate was only 80 and strong. His temperature and pulse chart is shown in *Fig. 213*. On Dec. 17 the tracheobronchial lymphatic glands were irradiated by the insertion of fifteen radon seeds of 1.5 mc. each by the method described by one of us (H. P. N.¹²). There was no reaction following this, and the patient left hospital on Dec. 23 with no cough and the wound healed.

On April 28, 1933, the patient stated that he was "feeling fine" and had gained two stone in weight since leaving hospital; he had no symptoms at all. Under the X-ray the right diaphragm was seen to be raised, and some movement was present on inspiration: the aorta was markedly dilated; in the right lung there was some thickening of the pleura and a shadow obscuring the right costo-phrenic angle. Fourteen radon seeds were seen in the mediastinum. The oblique view showed an indefinite opacity towards the posterior part of the right lung, which may represent consolidation breaking down in the centre.

PATHOLOGICAL REPORT.—On section the diseased lobe showed the appearance seen in *Fig. 214*. Microscopically this proved to be a squamous-celled carcinoma.

Case 9.—Jessie H., aged 19, machinist, was admitted to St. Bartholomew's Hospital on Nov. 26, 1932.

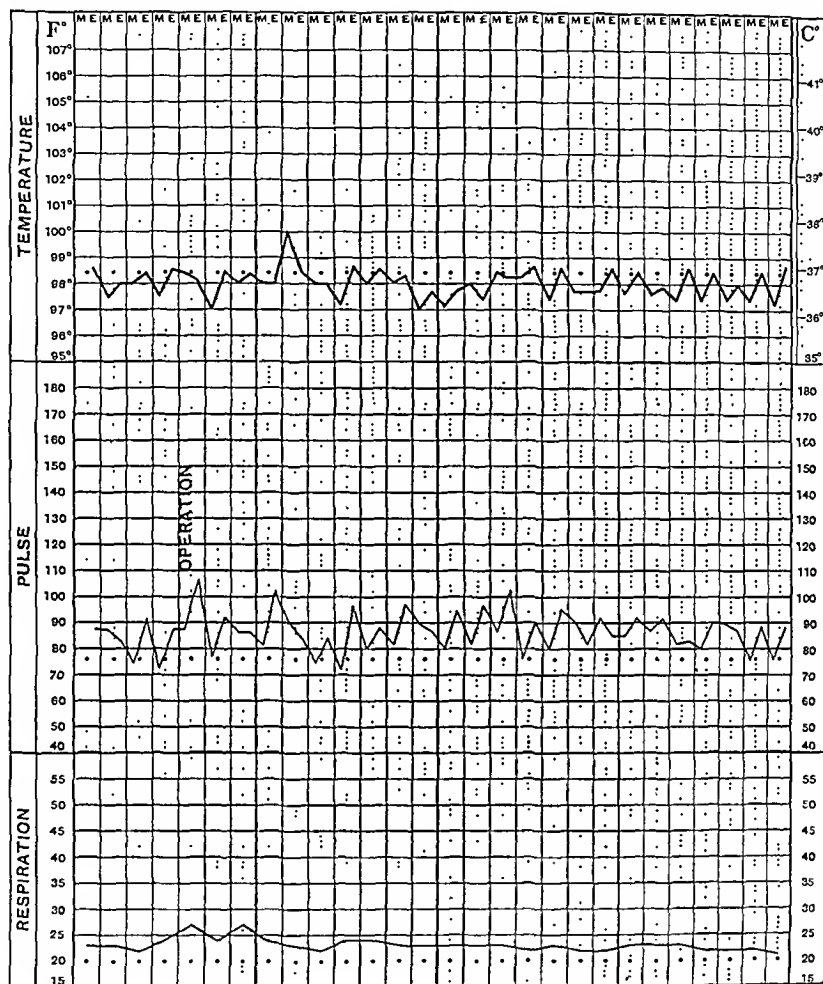


FIG. 213.—Case 8. Chart of the temperature, pulse, and respiration.

HISTORY.—There was a history of a cough and sputum for as long as she could remember. In January, 1932, she was admitted to the hospital for acute appendicitis, and following operation attention was drawn to her chest by the cough and copious sputum. There were signs of bronchiectasis at the left base, and lipiodol demonstrated a moderately severe degree of cylindrical dilatation in the left lower lobe. Nine months later she was readmitted to hospital as she still had 1 or 2 oz. of sputum daily, which was of foul taste and smell. On Nov. 28 the left phrenic nerve was exposed in the neck and crushed.

OPERATION (Nov. 30, 1932).—Left lower-lobe lobectomy. Anaesthetic (by Mr. Frankis Evans), nitrous-oxide-oxygen. Incision through the seventh interspace. The lower lobe was slightly smaller than normal, but the usual colour. There were adhesions surrounding the lower lobe, but only one or two over the costal

surface of the upper lobe. At the end of the operation the chest was drained by resection of a portion of the 9th rib. The operation lasted for exactly one hour. The blood-pressure at the beginning of the operation was 126/84 and the pulse-rate 84, while at the end of the operation the blood-pressure was 116/74 and pulse-rate 112.



FIG. 214.—Case 8. Section of the right lower lobe showing a squamous-celled carcinoma.

POST-OPERATIVE COURSE.—This was entirely uneventful. The closed drainage tube was disconnected on the fourteenth day, and the patient left hospital on Jan. 1, 1933, with normal temperature, pulse, and respirations, chest healed, and no sputum. On Jan. 6 the patient was feeling well and had no cough or sputum. On March 17 she was back at work; she had a slight cough, with $\frac{1}{2}$ oz. of sputum in the twenty-four hours. On examination of her chest there was poor movement at the left base but no added sounds could be heard in the lung. On April 7 she had a cough with only a trace of sputum, and was feeling quite well. On May 12 lipiodol filling demonstrated some bronchiectasis in the lower portion of the left upper lobe, which accounted for the persistence of sputum.

PATHOLOGICAL REPORT.—On section the diseased lobe presented an appearance almost identical with that seen in Case 5 (*see Fig. 209*).

Case 10.—Thomas S., aged 38, tram conductor, admitted to the Brompton Hospital on Nov. 17, 1932.

HISTORY.—He was quite well until July, 1926, when he coughed up a small clot of blood and was in bed for eight days with pain on the right side of the chest.

In December, 1927, he had several hæmoptyses of about 4 oz. followed by one of 20 oz. He spent ten weeks in hospital at this time and then had another severe hæmoptysis. From March, 1928, for two years he attended the Out-patient Department at one of the London chest hospitals, having frequent small hæmoptyses, and he was diagnosed as bronchiectasis. In 1932 he had six very severe hæmoptyses. Since 1928 he had had a small quantity—less than 1 oz.—of mucopurulent sputum. He had always felt well in himself.

ON EXAMINATION.—He was a tall, well-grown man, obviously very anæmic. General examination revealed nothing abnormal except at the right base behind, where there was a slightly impaired note and weak breath-sounds, with some post-tussive râles. The blood examination showed red blood-cells 4,500,000, hæmoglobin 66 per cent, white blood-cells 5000; the differential count was normal. X rays without lipiodol showed apparent bronchial dilatation at the right base; the left lung field appeared normal. After lipiodol, which unfortunately had entered the alveoli, some dilatation of the branches of the right descending bronchus could be seen. On Dec. 2 bronchoscopy showed some dilatation of the right main and middle bronchi. Although the cause of the hæmoptysis was still unknown, we considered that the seat of the trouble lay in the right lower lobe, and it was therefore decided to remove this lobe on account of the very severe bleeding. A pre-operative artificial pneumothorax was attempted but not obtained.

OPERATION (Dec. 10, 1932).—Right lower-lobe lobectomy. Anæsthetic (by Mr. John Hunter), nitrous-oxide-oxygen. There were a large number of firm adhesions over the lower lobe and a very well-marked fissure partly separating the apex of the lower lobe (the dorsal 'lobe') from the rest. After suturing the pedicle the end appeared to be so satisfactorily sealed with the lung tissue that the stump was not buried in the upper lobe on this occasion for the first time. The duration of the operation was one and a half hours, and at the end the condition was fair, but on return to the ward the patient was given a blood transfusion.

POST-OPERATIVE COURSE.—The patient's condition was on the whole satisfactory, but he ran a temperature up to 103° most evenings. The discharge from the chest soon became purulent, although the wound itself looked satisfactory. On Dec. 19 it was noted that he had 12 oz. of pus daily from the tube and was coughing up 1 oz. of sputum: the temperature was up to 102°. On Dec. 25 the patient suddenly collapsed, and it was found that the drainage bottle contained about one pint of blood. The drainage tube was immediately clamped and the patient was given a transfusion, and under nitrous-oxide-oxygen anæsthesia the chest was rapidly opened up. A basal empyema was discovered about 4 in. in diameter, some blood-clot was found in the cavity, and the end of the stump appeared necrotic with two bronchial fistulæ. There was a small vessel on the chest wall that was bleeding, but it was considered that this was probably the result of re-opening the chest and that the hæmorrhage had really come from the pedicle, although at the time the chest was opened (two hours after the bleeding) it had stopped. The cavity was packed with dry gauze and the wound left open.

When considering the cause of this man's empyema and secondary hæmorrhage we realized that this was the only case in which this calamity had occurred and was also the only occasion on which we had not buried the bronchial stump in the under surface of the upper lobe.

The infection in the wound gradually cleared up, and it became smaller, but in spite of this the patient's temperature continued. He complained of pain, and there was tenderness over the costal surface of his liver. An X-ray suggested a subphrenic abscess, which was confirmed by aspiration. Following the drainage of this abscess, the patient's temperature returned to normal. We consider that this subdiaphragmatic abscess resulted from injury to the diaphragm when dividing basal adhesions at the time of the lobectomy.

On March 18, 1933, the subphrenic abscess had healed. The patient started to get up, and was feeling very well. On April 7 he was discharged home to attend as an out-patient twice weekly. The chest wound was reduced to a sinus $2\frac{1}{2}$ in. deep and about 1 in. in diameter, with two minute bronchial fistulæ at the bottom

of the wound. There was practically no discharge from the wound; the patient had no cough, and there had been no recurrence of his hæmoptysis since the lobectomy. On May 12 the fistula and sinus were completely healed.

PATHOLOGICAL REPORT.—On section, the diseased lobe presented an appearance which we had not seen in any of the previous cases. One of the large tertiary bronchi was dilated in a saccular manner to the size of a primary bronchus: the wall of this dilatation was about 2 mm. thick and contained cartilage. The section of lung supplied by this bronchus was hard and fibrous, and contained many minute dilatations. The rest of the lower lobe, i.e., about nine-tenths of it, presented an entirely normal appearance.

SUMMARY OF CASES.

1. The *ages* of our cases varied from 13 to 55. Five cases were females and five males. The *predominant symptom* in six cases was cough and foul sputum, and in the other four a repeated hæmoptysis.

2. The *disease* for which lobectomy was performed was in two cases primary bronchial carcinoma and in eight cases bronchiectasis. Extensive pleural *adhesions* were present in seven cases, and practically none in three cases. *Lobes removed*: in seven cases the left lower, in two the right lower, and in one the right lower and middle lobes.

3. *Post-operative complications*: hæmorrhage in three cases, basal empyema in three cases, bronchopneumonia in one case, bronchial fistula in five cases (two of these were silent), and cerebral abscess in one case. The *average length of time* from operation to healing of the chest-wall sinus was five weeks.

4. *Present condition* (May 12, 1933):—

Dead (from cerebral abscess ten days after operation) ..	1 case
Dead (from influenzal pneumonia three months after operation)	1 „
Healed and free from symptoms	6 cases
Back at work, but with slight symptoms	2 „

CONCLUSIONS.

Although this series of ten cases is too short to justify the expression of results by percentages, it appears that by this technique the mortality of lobectomy may be considerably reduced, and that this operation will take its place as the treatment of choice in cases of unilobular bronchiectasis.

REFERENCES.

- ¹ SHENSTONE, N. S., and JAMES, R. M., *Canad. Med. Assoc. Jour.*, 1932, xxvii, 138.
- ² LILIENTHAL, H., *Thoracic Surgery*, 1925, ii. Philadelphia and London: W. B. Saunders Company.
- ³ ALEXANDER, J., *Surg. Gynecol. and Obst.*, 1933, lvi, 658.
- ⁴ SAUERBRUCH, F., *Die Chirurgie der Brustorgane*, 1925. Berlin: Julius Springer.
- ⁵ WHITTEMORE, W., *Ann. of Surg.*, 1927, lxxvii, 219.
- ⁶ ZAHLER, J. H., *Lancet*, 1929, i, 909.
- ⁷ HARRINGTON, S. W., cited by Ballou, Singer, and Graham, *Jour. of Thoracic Surg.*, i, No. 5, 537.
- ⁸ HEDBLUM, C. A., *Surg. Gynecol. and Obst.*, 1931, lxi, 406.
- ⁹ CORYLLOS, P. N., *Arch. of Surg.*, 1930, xx, 767.
- ¹⁰ BRUNN, H. B., *Ibid.*, 1929, xviii, 490.
- ¹¹ NELSON, H. P., *Lancet*, 1932, ii, 1003.
- ¹² NELSON, H. P., *Ibid.*, 1930, ii, 1118.

BRONCHIECTASIS :

A STUDY OF THE PATHOLOGY OF SIXTEEN SURGICAL
LOBECTOMIES FOR BRONCHIECTASIS.

By W. L. ROBINSON,

DEPARTMENT OF PATHOLOGY, UNIVERSITY OF TORONTO, AND THE TORONTO
GENERAL HOSPITAL CANADA.

THE problem of bronchiectasis, its etiology, pathology, and treatment, has held the interest and attention of clinician and pathologist alike for many years. Laennec¹ (1826) was probably the first to refer to this condition. The next important contribution to the subject was that of Corrigan² (1838), who, in a paper on cirrhosis of the lung, reported four cases. He considered the disease to be primarily a fibrosis of the lung with dilatation of the bronchi produced by traction of the scar tissue.

ETIOLOGY.

Congenital Factors.—As most of the early reported cases were noted in children, it was natural that a congenital factor should be emphasized as important in the etiology of this disease. This was first suggested by Grawitz³ (1880) when he reported a series of cases, mostly in children, all of which he believed were congenital in origin. Barlow,⁴ in the same year, reported a case in a child of 3 months. In the interior of the upper lobe of the left lung he found a cyst the size of a Spanish chestnut containing air, but with no apparent opening into it. Sauerbruch,⁵ who is probably the greatest exponent of this theory, believes that 8 per cent of the cases are congenital in origin. A congenital predisposition was thought by Duken⁶ to be important in some cases. Hedblom⁷ suggested the possibility of the development of the disease in a persistent fetal atelectasis when the chest cavity later expanded beyond the capacity of the atelectatic lung.

Mechanical Factors.—Mechanical factors, such as stenosis of the bronchi from foreign bodies in the lumina or from external pressure by aneurysm, enlarged glands, tumour growth, etc., have been emphasized by a number of writers (Meyer,⁸ Schott,⁹ Lemon,¹⁰ Hedblom,⁷ Biermer,¹¹ and Brunn and Faulkner¹²). It has, however, been generally admitted by these men that an added infection of the bronchial wall was a necessary concomitant. Such a mechanical factor as increased intrabronchial air-pressure in coughing is given by Schneider¹³ as sufficient cause in itself to produce one type of bronchiectasis, viz., the atrophic type. Biermer¹¹ thinks the mechanical pressure of retained secretions in the lumen added to an increased intrabronchial air-pressure is important.

Mechanical stresses such as lateral pulls on the walls of the bronchi from fibrosis of the lung parenchyma, pleural adhesions, collapse of the lung, etc., are mentioned as causes in some cases, (Jex Blake,¹⁴ Coplin,¹⁵ Coats,¹⁶ Brooks,¹⁷ Hedblom,⁷ McFarland,¹⁸ and Kaufmann¹⁹). Hedblom says that atelectasis in adults may produce bronchiectasis by virtue of an increased negative pleural pressure, lessening as it does the support to the outside of the bronchial wall and allowing the atmospheric pressure to produce its dilating effect. More recently Warner,³⁸ studying bronchiectasis from a clinical and radiological standpoint, laid stress upon the mechanical influence of increased negative intrathoracic pressure occurring in cases of lobar atelectasis. He has shown that bronchiectasis may develop within a few weeks of the onset of a massive collapse or atelectasis. Infection, however, he believes, precedes these changes, producing a weakening of the walls of the larger bronchi and a swelling of the mucosa with obstruction of the bronchioles.

Infection.—Infection of the bronchial wall is generally accepted by all as being of most importance. Graham and Warner,³⁹ in their study of bronchiectasis associated with triangular shadows, conclude that "Infection is the all-important factor". Its relation to chronic sinus disease is emphasized by Lemon,¹⁰ Webb,²⁰ and Adam,²¹ the sinuses being the primary source of infection. A chronic infective process in the wall of the bronchus, however, is considered by others as being a complication or sequela of such diseases as measles, whooping-cough, bronchopneumonia, and acute bronchitis (Schneider,¹³ Lemon,¹⁰ Jex Blake,¹⁴ Hedblom,⁷ Klare and Reusse,²² McNeil et al.,²³ and Findlay and Graham²⁴). The infection, Meyer⁸ thinks, is probably due to a loss of antiseptic properties of the bronchial mucus. The retention of copious secretions is given also as a factor by Meyer and by Biermer.¹¹ Specific types of infection are not mentioned by these authors. Smith,²⁵ however, from the observations of many clinical cases and the study of much experimental material, concludes that primary bronchiectasis is due to a specific type of infection, namely, fuso-spirochætal infection.

Other Factors.—Other factors have been suggested, such as loss of nerve control of the bronchial musculature (Lebert²⁸ and Biermer¹¹), overwork of the bronchi (Black²⁹), and nutritional disturbances from an endarteritis of the bronchial arteries (Brunn³⁰). In reference to the latter, Sauerbruch and Brunn did some experimental work on the tying of the branches of the pulmonary artery. They were able thereby to produce a fibrosis the contraction of which caused bronchiectatic dilatations of the bronchi of the affected lobe. Duken⁶ and Ochsner²⁶ believed that in the earlier phases bronchiectasis may be the result of a loss of tone in the muscular wall and the inability of the bronchus to empty itself of secretions.

ANATOMY AND PHYSIOLOGY.

Anatomy.—The bronchial walls vary slightly in structure from the bifurcation of the trachea to their terminations in the lung parenchyma. Throughout their course they are lined by a ciliated type of columnar epithelium supported by a stroma with a number of mucous glands opening on the surface. Macklin³¹ has shown that the elastic elements are distributed

in longitudinal as well as circular bundles. There are several layers, the most conspicuous being situated in the mucosa and running longitudinally. They are branched and grouped in fasciculi, allowing thus for dilatation of the bronchi as well as lengthening. Just outside of the longitudinal elastic membrane of the mucosa is another layer running mainly circularly. These latter fibres are interwoven with the muscle bundles. Other layers of lesser importance are also present.

The bronchial musculature extends throughout the whole length of the air-passages (Baltisberger³²). In the main branches, where the cartilage plates are distributed around the entire circumference of the wall, the muscle fibres form a complete layer located between them and the mucosa. The muscle bundles, while in the main encircling the tube, branch off diagonally to form a more or less continuous network in 'lazy-tong' fashion, or, as Miller³³ expressed it, run in "geodesic lines". The walls of the main branches down to the point where they have a diameter of 0.5 mm. are reinforced by cartilage plates, which prevent them from collapsing on inspiration.

Physiology.—Macklin³¹ has shown that the bronchi during inspiration both widen and elongate. During expiration they contract and shorten. Whether this is done simultaneously throughout the whole tubular system or passes as a wave from larynx to periphery of lung and vice versa is not known. He suggests, however, that a contraction of the bronchi passing along as a wave from periphery to hilus would obviously best serve in emptying the lung of air.

These points in reference to the anatomical structure and physiology of the bronchial system serve to show that it is not a rigid system of tubes operating passively as conduits for the passage of air between the larynx and lung alveoli, but a very actively functioning part of the respiratory system, dilating and lengthening during inspiration to accommodate its many branches to the inrush of air and the expansion of the lung, and then conversely contracting and shortening probably in a wave-like peristaltic movement during the process of expiration. During shallow breathing, of course, this is slight in amount, but becomes considerable with coughing or other violent respiratory movements. The ability of the bronchi to return to a normal diameter depends, as in blood-vessels, upon the integrity of the muscle and elastic elements. Any damage to these structures must of necessity lead to a loss of resiliency and contractile power in the unduly dilated wall. It is this failure to return to the normal state of rest which eventually leads to the permanent state of relaxation and dilatation of bronchiectasis.

PATHOLOGY.

Bronchiectasis is considered as a permanent dilatation of one or more bronchi. Temporary dilatations which have been demonstrated radiologically no doubt are due to weakness and loss of tone of the bronchial muscles continuing over a relatively short space of time. This probably occurs in the early stages of the development of the disease, and if no disintegrative changes take place in the bronchial wall, complete restoration is quite possible. Later with irreparable damage to the essential structures of the wall, particularly

the musculature and elastic elements, a return of the bronchi to their normal diameter is impossible, and dilatations become inexorably fixed. The etiological factors leading to this latter condition may be numerous, but the essential pathological lesion, I believe, is the same in all cases—namely, a weakening or loss of integrity of the musculo-elastic system.

Within the last two years we have had the opportunity of studying, both bacteriologically and pathologically, a series of sixteen cases of bronchiectatic lesions in lobes of lungs freshly removed surgically for intractable symptoms of bronchiectasis. These were removed from patients of both sexes, with ages varying from 14 years to 52 years. A clinical report of this series of cases is presented by Dr. Janes on pp. 273-5. A previous communication by Shenstone and Janes³⁷ covers in part this same series. The lobe of lung when removed by the surgeon was immediately brought to the laboratory in sterile towels, and here with sterile instruments the affected bronchi were opened and cultures of various types made. The bacteriological findings have been reported in another paper by Dr. Greey.³⁵ He found in a careful study of nine of the cases that there was no uniform bacterial flora. Spirochaetes were found in four of the cases, and pure cultures of streptococcus in three of them.

The lung specimens were then trimmed and prepared for mounting. Blocks were taken from various areas along the course of the dilated bronchi. These were placed immediately in Zenker's fluid or in 10 per cent formalin. Transverse and longitudinal sections were made, and stained with hæmatoxylin and eosin, Van Gieson, Mallory's phosphotungstic acid, hæmatoxylin, and Masson's stain, and a combination of Masson's and Weigert's elastic tissue stain. This latter combination gave very excellent results. Preparations with Gram-Weigert's stain for bacteria and Levaditi's silver stain for spirochaetes were also made.

The important feature of this series is the fact that the material was obtained in a fresh state and in some cases fairly early in the development of the disease. An opportunity was thus afforded to study the evolution of the disease process from the preliminary inflammatory reaction to the final fully developed permanently dilated bronchial tube. The lobes of lung removed at operation were all lower ones, including in some cases a portion of the upper lobe, and from both right and left lungs. The dilatations were of the fusiform or cylindrical types and varied from 4 mm. to 1.5 cm. in diameter (Fig. 215). The secondary branches were most frequently involved. The mucous membrane was of a peculiar pinkish-yellow colour, and was soft.



FIG. 215.—Bronchiectatic lung removed from a female, aged 21 years, with a history of cough since childhood.

succulent, almost redundant in appearance. The lumina for the most part contained in their proximal portions very little secretion. This was either yellowish and mucoid in character or yellowish-grey and inspissated. The finer bronchioles leading off from the dilated proximal portions were, however, usually well filled with a yellowish mucoid material. The thickness of the walls varied, as a rule being definitely thicker than normal. In the vast majority of them there was little gross evidence of involvement of neighbouring lung tissue beyond a variable amount of lung collapse. In none was there any evidence of tuberculosis.

Histopathology.—An analysis of the histological preparations revealed findings that were fairly constant throughout. A definite inflammation of the wall was found in all cases, with degenerative changes in certain essential

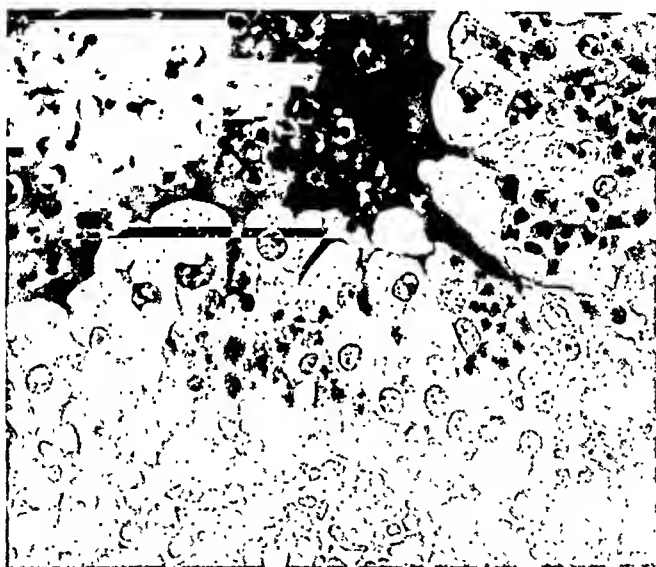


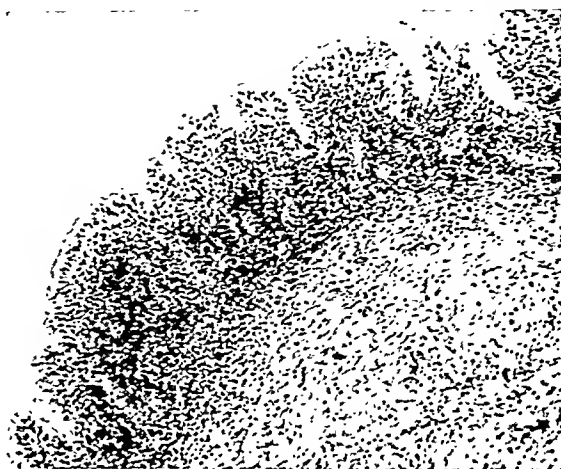
FIG. 216.—Lining cells are swollen, irregular in size and shape, and cilia are for the most part gone. ($\times 400$.)

structures added thereto. The reaction was, however, essentially an inflammatory one. In the more acute cases the beginnings of the process were apparent. The more advanced processes were seen in the longer-standing cases. The more acute and early cases showed a marked hyperplastic state of the mucosal epithelium. The cells were large, swollen, irregular in outline and size, and tended to grow up in a polypoid manner, increasing thereby the thickness of the mucosa. In all but two, however (acute cases), the cilia of the lining epithelial cells were intact. In these two cases the lining cells were swollen, irregular in size and shape, and the cilia for the most part gone. This is illustrated in *Fig. 216*, a case with a three and a half months' history. Considerable quantities of exudate, consisting of mucus with large numbers of polymorphonuclear leucocytes, occluded the lumina of the smaller branches. In view of the frequency of hæmoptysis in these cases it was

interesting to note that very few ulcers of mucosa were found. These occurred only in the more acute cases and were microscopic in size.

The most striking changes, however, were found in the subepithelial layer. Here, lying between the lining epithelium and the cartilage plates,

FIG. 217.—Inflammatory infiltration of the subepithelial layer. ($\times 80$.)



are found important structures—namely, the bronchial musculature and elastic fibres. In this zone a most marked inflammatory reaction occurred (*Fig. 217*). The inflammation was somewhat granulomatous in character, the growth



FIG. 218.—Degenerative changes in muscle fibres and some splitting of the elastic lamellæ are shown. ($\times 160$.)

of new blood-vessels and connective tissue being a prominent part of the process. In the earlier stages, as seen in the acute cases, this granulomatous feature was not so apparent, and the muscle and elastic fibres were found for the most part intact. Here and there, however, beginnings of a degenerative

change were seen in the muscle fibres and some splitting of the elastic lamellæ was also evident (*Fig. 218*). A moderate infiltration of lymphocytes and plasma cells permeated these tissues and some œdema was present. One frequently saw evidences of weakening of the wall in the outpouching of the

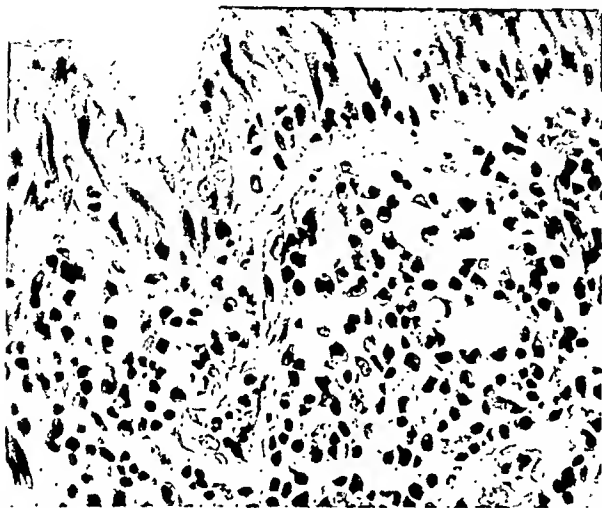


FIG. 219.—Section of mucosa showing character of the subepithelial inflammatory reaction. ($\times 320$.)

mucosa at the points where the muscle and elastic tissues were destroyed. The capillaries of the mucosa stood out prominently, the endothelial layer being made up of large succulent cells. They appeared to rise abruptly in the outer layers and to run at right angles directly to the lining epithelium. In this stage very little increase of connective tissue stroma was seen.

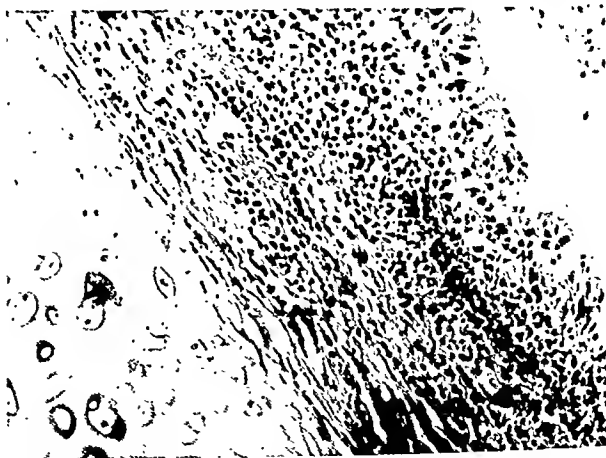


FIG. 220.—Muscle and elastic elements replaced by fibrous or granulation tissue. ($\times 160$.)

In the longer-standing cases no particular change in the character of the inflammatory infiltration was apparent (*Fig. 219*). Destruction of muscle and elastic elements, however, was quite pronounced, and in many bronchi was complete. They were replaced by a fibrous or granulation tissue (*Fig. 220*)

and densely infiltrated with lymphocytes and plasma cells. The capillaries stood out prominently, being lined by one or more layers of large swollen endothelial cells. A peribronchial inflammation became apparent, with some

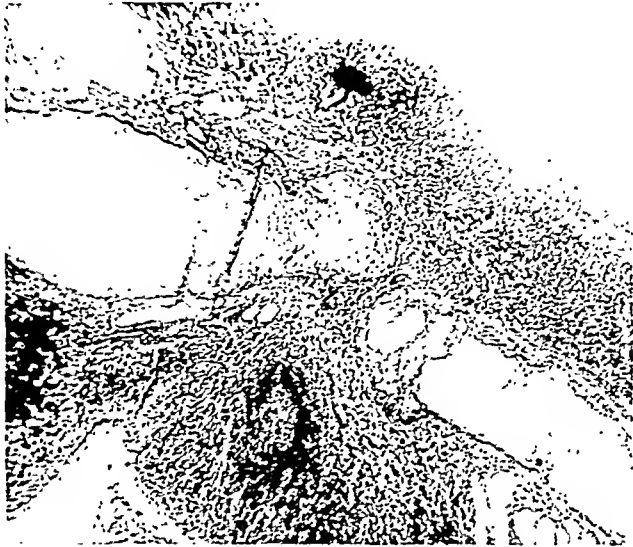


FIG. 221.—Involvement of cartilage plates leading to destruction and replacement by fibrous tissue. ($\times 48$.)



FIG. 222.—Intimal thickening of wall of bronchial artery producing the endarteritis obliterans type of arteriosclerosis. ($\times 160$.)

fibrosis of a narrow zone of the adjacent lung parenchyma. The involvement of the cartilage plates at times was rather extensive, leading to destruction and replacement by fibrous tissue. This is illustrated in *Fig. 221*, where the

process has involved the septa between two bronchi, tending to a melting down of the intervening wall. In the rest of the cases, where the process was more definitely established, it was interesting to note that the lining epithelium was intact throughout, and consisted of normal stratified columnar epithelial cells with well-defined cilia. Fresh scrapings were made from some of these and examined with dark-field illumination. The cilia were observed to continue their whipping movements for some hours after removal.

A feature of the process that perhaps has not been sufficiently emphasized in the past was the involvement of the bronchial arteries. A definite intimal thickening with stenosis of the bronchial arteries was found in 65 per cent of the cases. These lesions were found only in the bronchial arteries and only in those of the dilated bronchi. The thickenings of the walls were intimal and therefore of the type of endarteritis obliterans. This is illustrated in *Fig. 222*. Whether or not this was consequent upon the inflammation of the bronchial wall, or an etiological factor, is difficult to determine. While it may not have been of prime importance, it is quite possible that it later played a part in diminishing the blood-supply and lessening the resistance of the tissues. An example somewhat akin to this is seen in the experimental work of Hepler³⁶ in the production of solitary cysts of the kidney by the combined interference with circulation and obstruction to the urinary outflow.

COMMENT.

The outstanding and most consistent pathological finding in our series was a chronic inflammatory condition of the bronchial walls with various degrees of damage up to complete destruction of the musculo-elastic tissue. Infection of the wall would therefore seem to be of prime importance in the development of this disease. In the early stages the condition is probably of the nature of a paresis of the muscular system leading to loss of tone with consequent dilatation. Provided the infection is of short duration, recovery at this stage might be complete. When, however, the infection has persisted for a longer period of time permanent damage occurs, the resiliency of the wall is lost, and the dilatations become fixed. With the dilatation, even if temporary, a vicious cycle is established: Bronchial secretions tend to accumulate, become infected, with spread to the adjacent walls. The damaged walls then slowly give way under the intermittent strain of ordinary or violent respiratory movements. There was nothing found in our series of cases to indicate that mechanical overstrain such as pleural adhesions, collapsed lung, etc., had played a part.

Mechanical obstructions such as an aspirated foreign body, tumour growth, etc., are found in certain cases. None of our cases, however, revealed such conditions. Where present they play their part, I believe, by causing a local lowering of resistance of mucosa and blocking of normal secretions. Infection then becomes established within the lumen of the bronchus, and from here soon spreads to the wall, involving the whole bronchial tree distal to the obstruction. Here it persists and leads to degenerative changes in the musculo-elastic elements of the wall.

The vascular changes occurring in the bronchial arteries, if not a prime factor in the development of this disease, are no doubt an important contributory one, in that they lower the resistance of the tissues and favour persistence of the infection.

In cases where no mechanical obstruction is present such a thing as a physiological block to the clearing out of secretions might conceivably occur. A persistent local infection at the orifice of one branch may damage the ciliated epithelium to such an extent that the cilia become paralysed and the mucus in the distal portion accumulates to pathological proportions. A metaplasia of epithelium to a squamous-celled type at this point would have the same



FIG. 223.—Metaplasia of bronchial mucosa from columnar to squamous-cell type. (\times 160.)

effect. Fig. 223 illustrates this latter type of block. The section was taken from the proximal end of the bronchiectatic tube. In a more recent case, not included in this series, I found all the dilated bronchi in the affected lobe to be lined with squamous epithelium. In such cases there must be a definite physiological block to the clearing of secretions from the more distal portions of the bronchial tree. Following the stagnation of secretions, and infection, a spread through the bronchial wall would lead to the same chain of events and terminate in bronchiectatic dilatations consequent upon a damage to the muscle and elastic elements.

REFERENCES.

- ¹ LAENNIC, "De l'Auscultation médiate", 1826, quoted by McNeil, *Arch. of Dis. Child.*, 1929, iv, 170.
- ² CORRIGAN, *Dublin Med. Jour.*, 1838, xiii, 266.
- ³ GRAWITZ, P. von, *Virchow's Arch.*, 1880, lxxxii, 217.
- ⁴ BARLOW, M., *Brit. Med. Jour.*, 1880, Jan.
- ⁵ SAUERBRUCH, *Wien. klin. Woch.*, 1927, xl, 543.
- ⁶ DUKES, J., *Zeits. f. Kinderheilk.*, 1927, xlv, 1.
- ⁷ HEDBLUM, C. A., *Surg. Gynecol. and Obst.*, 1931, lxi, 406.

- ⁸ MEYER, WILLY, *Trans. Amer. Surg. Assoc.*, 1914, xxxii, 592.
- ⁹ SCHOTT, E., *Dent. med. Woch.*, 1926, lii, 1258.
- ¹⁰ LEMON, *Med. Clin. N. Amer.*, 1926, x, 111, 531.
- ¹¹ BIERMER, *Arch. f. pathol. Anat.*, 1860, xix, 241.
- ¹² BRUNN and FAULKNER, *Amer. Rev. Tuberc.*, 1929, xix, 191.
- ¹³ SCHNEIDER, H., *Beitr. z. pathol. Anat.*, 1928, lxxix, 446.
- ¹⁴ JEX BLAKE A. J., *Brit. Med. Jour.*, 1920, May.
- ¹⁵ COPLIN, W. M., *Manual of Pathology*, 1910. Philadelphia: P. Blakiston's Son & Co.
- ¹⁶ COATS, J., *A Manual of Pathology*, 1900, 4th ed. London: Longmans, Green & Co.
- ¹⁷ BROOKS, H. T., *Text-Book of General and Special Pathology*, 1912. Philadelphia: F. A. Davis Co.
- ¹⁸ MCFARLAND, J., *Text-Book of Pathology*, 1910. Philadelphia: W. B. Saunders Co.
- ¹⁹ KAUFMANN, E., *Pathology*, 1929. Philadelphia: P. Blakiston's Son & Co.
- ²⁰ WEBB, A. G., *Jour. Amer. Med. Assoc.*, 1921, lxxvi, 714.
- ²¹ ADAM, J., *Jour. Laryngol. and Otol.*, 1925, xl, 172.
- ²² KLARE and REUSSE, *Beitr. z. Klin. d. Tuberk.*, 1926, lxxiii, 255.
- ²³ McNEIL et al., *Arch. of Dis. Child.*, 1929, iv, 170.
- ²⁴ FINDLAY and GRAHAM, *Ibid.*, 1927, ii, 73.
- ²⁵ SMITH, D. T., *Arch. of Surg.*, 1930, xxi, 1173.
- ²⁶ OCHSNER, A., *Amer. Jour. Med. Sci.*, 1930, clxxix, 388.
- ²⁷ FISHER, W., *Ziegler's Beitr.*, 1889, v, 453.
- ²⁸ LEBERT, quoted by Ochshner, *Amer. Jour. Med. Sci.*, 1930, clxxix, 388.
- ²⁹ BLACK, C., *Monthly Jour. Med. Sci.*, 1853, xvi (Edinburgh).
- ³⁰ BRUNN, *Arch. of Surg.*, 1929, xviii, 490.
- ³¹ MACKLIN, C. C., *Physiol. Reviews*, 1929, ix, 1; *Arch. of Surg.*, 1929, xix, 1212; *Amer. Rev. Tuberc.*, 1932, xxv, 111.
- ³² BALTISBERGER, W., *Zeits. f. Anat. u. Entwickl.*, 1921, lxi, 249.
- ³³ MILLER, W. S., *Amer. Rev. Tuberc.*, 1921, v, 689.
- ³⁴ SACRERBRUCH and BRUNN, quoted by Meyer, *Trans. Amer. Surg. Assoc.*, 1914, xxxii, 592.
- ³⁵ GREY, P., *Jour. of Infect. Dis.*, 1932, l, 203.
- ³⁶ HEPLER, A. B., *Surg. Gynecol. and Obst.*, 1930, l, 668.
- ³⁷ SHENSTONE, N. S., and JAMES, R. M., *Canad. Med. Assoc. Jour.*, 1932, Aug., 138.
- ³⁸ WARNER, W. P., *Ibid.*, xxvii, 583.
- ³⁹ GRAHAM, D., and WARNER, W. P., Personal communication.

ETIOLOGY AND TREATMENT OF SLIPPED EPIPHYSIS OF THE HEAD OF THE FEMUR.*

By E. N. WARDLE, LIVERPOOL.

It is proposed to divide this paper into two sections: (1) A consideration of the etiology of the condition; (2) A description of a new method of treatment, with detailed reports of three consecutive cases in which it has been used successfully; followed by a critical survey of the methods employed by other writers, and their recorded results, leading up to the basis on which the present method was evolved.

The clinical findings are well recognized and accurately described in various writings quoted. *Case 9* of this series was included because it has some bearing on the question of etiology; but it also serves to emphasize one clinical point which is not stressed in the literature—namely, that the signs in this condition are those of a fractured neck of the femur.

The X-ray pictures and their significance are a subject on which there seems to be agreement in general, but a further consideration of this is included later.

ETIOLOGY OF SLIPPED EPIPHYSIS.

First of all a summary of the cases examined, from the etiological standpoint.

Case 1.—J. R., male, aged 15 years. First seen on July 2, 1931, with a history that a month earlier he had fallen off a bicycle and *struck the left knee*. A week after this he began to have pain in the left hip and was unable to bear weight on the limb. Overweight and overheight for his age. (For details, see account in second section.)

Case 2.—H. Ma., male, aged 13 years. First seen on April 23, 1931, complaining of a limp. No pain and no history of trauma was elicited. No signs of endocrine disturbance. (Fig. 224.)

Case 3.—C. R. W. H., male, aged 14 years. First seen on Nov. 22, 1931.



FIG. 224.—*Case 2.* X-ray showing (X) areas of rarefaction in metaphysis.

* A preliminary article on work undertaken for the Lady Jones Research Fellowship in the University of Liverpool.

Had noticed a little pain in the hip one month previously. One week before had knocked left ankle while going upstairs, and the day afterwards could not walk on the left leg. (For details, see second section.)

Case 4.—H. Mo., male, aged 7 years. No history available.



FIG. 225.—*Case 5.* Right hip. Epiphysis displaced and rarefaction of metaphysis still apparent.

FIG. 226.—*Case 5.* Knees, showing metaphysal expansion and rarefaction in both epiphysis and metaphysis.



Case 5.—W. P., male, aged 16 years. First treated for genu valgum in January, 1930, but reported back to hospital in September, 1931, with 'scissor-leg' deformity, and X rays at this time showed generalized epiphysal involvement, including slipping of one upper femoral epiphysis. (Figs. 225–230.)

FIG. 227.—*Case 5.* Involvement of lower epiphyses of tibia and fibula.

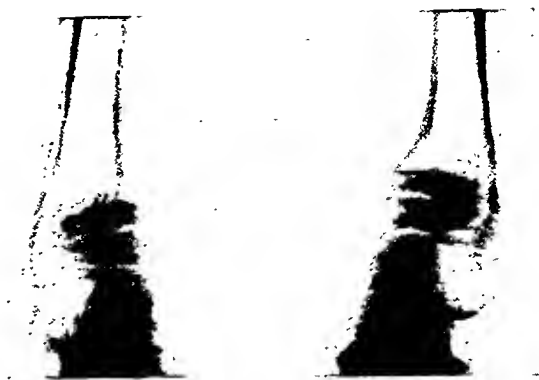
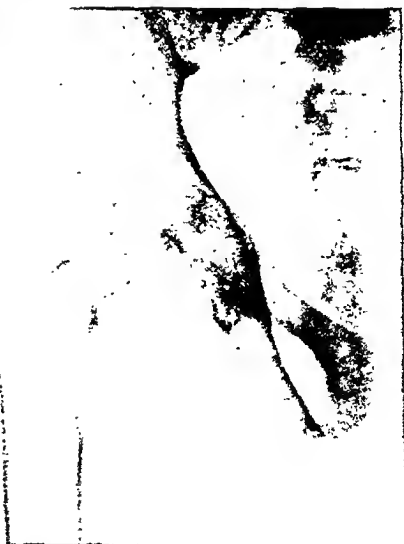


FIG. 228.—*Case 5.* Metaphyseal expansion and rarefaction in wrists.

FIG. 229.—*Case 5.* Left hip. Marked rarefaction in metaphysis, but displacement of epiphysis is only just commencing.



Case 6.—M. S., female, aged ? years (since dead). X rays show generalized epiphysial involvement, and a diagnosis of renal rickets was made.



FIG. 230.—*Case 5.* Changes in upper end of right humerus similar to those already shown in right femur.

Case 7.—S. J., male, aged 17 years. History of a fall in September, 1931, in which the right thigh was wounded by a spike. This healed. In November he



FIG. 231.—*Case 7.* X shows large area of rarefaction in metaphysis.

started to have pain in the right thigh and hip, and by December was unable to bear weight on the limb. (*Fig. 231.*)

SLIPPED EPIPHYSIS OF HEAD OF FEMUR 317

Case 8.—R. D., female, aged 4 years. Complete separation of epiphysis of the head of the femur by direct crushing injury. Epiphysis extruded from out of the acetabulum. (Fig. 232.)



FIG. 232.—*Case 8.* X shows outline of capital epiphysis, extruded from the acetabulum on to the dorsum ilii. Note that the relation of the greater trochanter to the neck remains unchanged.

Case 9.—J. D., male, aged 13 years. Following an injury to the hip, a direct blow on greater trochanter, he was found to lie flexed, adducted, and everted at the hip-joint and to have $\frac{1}{2}$ in. real shortening. A diagnosis of slipped epiphysis was considered, but X rays showed a fracture through the trochanteric region (Fig. 233).

Case 10.—D. L., female, aged 15 years. Four years previously this patient had had a trans-trochanteric osteotomy, and X rays suggest a slipped epiphysis. It was interesting to note that on Jan. 13, 1932, she had 1 in. real shortening, $\frac{1}{4}$ in. apparent shortening, and that she could abduct 45° at the hip-joint; she had half normal range of rotation, a compensatory left dorsal scoliosis, and walked well without a noticeable limp.



FIG. 233.—*Case 9.* Showing fracture through the trochanteric region.

Case 11.—A. G. H., male, aged 17 years. First seen on April 13, 1933. He had noticed that he was walking with a limp for eight weeks previously and that his left knee and ankle 'gave way' easily. There was no history of any previous accident or illness. (For details see second section.)

According to the literature there is no general agreement on the question of etiology. While the factors are agreed upon, their relative value is not. From 1907, when Elmslie¹ wrote upon the subject, until 1926 there had been no notable additions to the literature. In that year Key,² in an article which contained a considerable amount of original work, also included an excellent résumé of previous writings, and summed up the position with regard to

etiology. He maintained that there were three important etiological factors: (1) Trauma; (2) Static forces; (3) Endocrine disturbance; and that while it could be shown that each of these played a part he was unable to assess their relative value, and stated that the three of them were never present together. Other writers since then have either followed Key or evaded the vexed question of etiology altogether.

Badgley² (1929) emphasizes Key's statement that the periosteum around the neck of the femur becomes thinner during adolescence and that the capital epiphysis is thereby deprived of support; he also records the fact that the plane of the epiphysial line tends to become more vertical at the start of the second decade.

Willis³ (1929) leaves etiology with the statement that "the majority of his cases have occurred following definite injury in overweight adolescents of the infantile type." This includes all three accepted factors and implies that they may all operate in the same case.

Irwin⁵ (1929) mentions thinning of the periosteum and alteration of the plane of the epiphysial line towards the vertical, but does not agree with either of them. He states that up to ten years of age the cartilages of head and greater trochanter are connected and that it is wellnigh impossible to separate one, without the other, by force. This seems to imply a belief in trauma as the main etiological factor. Case 8 of this series is interesting from this point of view, because in it the head of the femur in a child aged 4 was forced out of the acetabulum and the greater trochanter left behind in the normal situation (*see Fig. 232*).

Jahss⁶ (1931) makes no statement on the question of etiology. Perkins⁷ (1932) points out that the flake of diaphysis, usually separated with other epiphyses, is not seen in the case of the head of the femur. This is true for all the X-rays studied, but, in the writer's opinion, cannot be used as evidence to rule out trauma from the etiology: it merely indicates that the degree of force producing the lesion in the hip is much less than that which displaces other epiphyses. It is definite evidence, however, that there is normally a secure attachment between epiphysial cartilage and metaphysis, and suggests that there is some weakening of this attachment, in the case of the head of the femur, before the displacement commences.

With regard to my cases, in four of them I was able to verify the history of trauma from the patient's parents or from other responsible people, and in each one of these there was a definite history of an *indirect* injury, i.e., the force had been applied to the knee or foot. It is interesting to contrast these with Cases 8 and 9, in which there was an equally definite history of direct injury to the region of the hip-joint and in neither of which did a typical slipped epiphysis occur. I feel that this fact is of importance and is of help in advancing an hypothesis of the etiology.

Two cases had generalized epiphysial involvement, and the X-ray findings were consistent with what has been termed 'renal rickets', and the patients were in the right age group.

It seems reasonable to suppose that a young patient would easily forget a mild trauma to the knee when questioned regarding the hip. From a consideration of these facts I have come to the conclusion that, with

SLIPPED EPIPHYSIS OF HEAD OF FEMUR 319

regard to the etiology, cases of slipped epiphysis can be divided into two types:—

1. Those with definite evidence of disordered internal secretion in which there will be other epiphyses involved besides that of the head of the femur.

2. Those with a history of indirect trauma, however small, where only the capital epiphysis of the femur is implicated and where there are no other signs of disease.

Once the epiphysis has 'gone over the brink', it seems reasonable to conclude that body weight will have a very definite effect in increasing the deformity, and this is probably the explanation of Key's statement that the three factors are never found together.

The 'overweight' type of case, so frequently referred to in the literature, but not so often seen, should be placed in the second class, with emphasis on the fact that the increased body weight is a secondary factor. Most of the recorded cases are unilateral.

It remains to consider whether there is any connecting link between these two types, and if so, what the nature of it is.

The kind of injury, and the interval between it and the onset of the symptoms and signs, which is noteworthy in the cases cited, suggests the tearing of periosteum and blood-vessels rather than damage to bone or cartilage. The natural consequence of such damage would be a reactive hyperæmia. Further, the acetabulum grips the head of the femur so firmly that any strain applied must fall on or near the region of the epiphysal line.

In bone, hyperæmia produces decalcification (Leriche and Polichard⁸), and I think evidence of rarefaction is to be seen in the early X-rays of these cases, at a time when it cannot be accounted for by immobilization, in the metaphysis. In rickets and in 'renal rickets' it is known that decalcification occurs in the metaphysal and epiphysal regions.

We are therefore brought back to the statement of Kocher⁹ in 1894, quoted by Key, Irwin, and de Quervain,¹¹ that the underlying factor in slipping of the femoral epiphysis is a 'localized osteomalacia' of the neck of the femur. The decalcification occurs in the area of most recently formed bone and thus weakens the attachment of epiphysal cartilage to metaphysis.

As mentioned above, there is no flake of diaphysis detached with the epiphysis because the original force is small and does not produce any displacement of itself.

The explanation of the cases quoted as having no history of injury is that the injury was slight and not considered worthy of notice by the patient. It is interesting to note that in the older literature up to 1907 this condition is always referred to under the heading of 'traumatic coxa vara', although the authors Kocher,⁹ Kirmisson,¹² and Elmslie¹ all emphasize the small amount of force recorded in the case histories. It seems that later writers, impressed by the minimal trauma, decided for that reason alone that the injury was of no significance in the etiology.

Either trauma or endocrine disturbance, therefore, produces a weakening at the metaphysis which allows the epiphysis to slip gradually under the influence of either body weight or muscular action.

The three factors are, therefore, accounted for, and the reason why they are never all present together is apparent—body weight cannot act until the process has been started by one of the other factors.

TREATMENT OF SLIPPED EPIPHYSIS.

The first stage consists in placing the patient on a well-made and accurately fitted frame (*Fig. 234*) which is constructed in the manner of a Jones spinal frame with extension ends to the leg pieces. Studs are provided on the pelvic bars for the attachment of a groin strap. It will be noticed that no provision is made for abduction of the lower limbs.

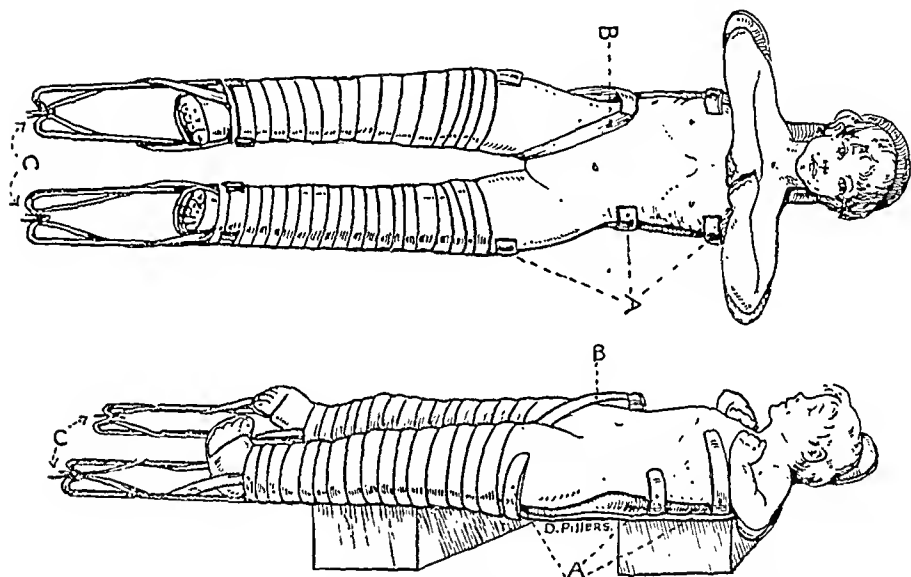


FIG. 234.—Illustrating position of patient on frame. A, Side bar of frame; B, Groin strap on side of lesion; C, Extension ends, with tapes tied. Note blocks, which facilitate nursing.

Strapping extensions, with tapes attached, are then applied to both legs from the groins to the malleoli, as used for fixed traction in a Thomas bed knee splint. A groin strap is now fixed *on the side of the lesion*, by means of two studs riveted to the side wing of the frame. Traction is then applied by tying both sets of extension tapes over the extension ends of the leg-pieces, that on the sound side being used to steady the pelvis. Finally both limbs are bandaged to the frame, and on the affected side this bandage is used gradually to correct the external rotation which is generally found to be present.

It will be seen that the result is steady and increasing traction applied to the affected hip in a relatively adducted position, i.e., the shaft and neck of the femur are being pulled in an axis parallel to the vertical axis of the body, while the groin strap, and traction on the unaffected leg, provide two counter-balancing forces which prevent both adduction and abduction at the injured hip-joint.

A most important part of the treatment is the nursing, and it is a steady application of simple routine principles which is necessary. All pressure points must be rubbed with spirit until dry, and then powdered, once every four hours day and night, and extension tapes tightened at the same time. Particular attention must be paid to the groin from which counter-pressure is being taken. Control X-rays are taken at intervals of fourteen days until the epiphysis has slid back into place, and traction is continued for an arbitrary period of one month after this has happened.

For the second stage the patient is removed from the frame but is kept recumbent and is allowed free *active* non-weight-bearing exercises at the hip-joint. This is continued until the range of movement no longer increases, and, as will be seen from the case reports, no definite period can be allotted. But as the patients are young, and time is of relative unimportance, it is much better to prolong the period and make certain that the reposition has become stable.

In the last stage of treatment the patient is allowed up in a walking caliper splint, and this must be worn for at least one year from the time that the treatment was commenced. Exercises in recumbency may be continued with advantage.

Case 1.—J. R., male, aged 15 years. First seen on July 2, 1931, and gave the history that a month earlier he had fallen off a bicycle and struck his left knee on the ground. One week after the accident he commenced to have pain in the left hip and became unable to stand on the affected leg. It was noted that his general condition was good but that he was definitely overweight and overheight for his age.

July 2, 1931.—X-ray shows condition of left hip (*Fig. 235*). He was placed on a frame in the manner described.

July 23.—X-ray shows partial reduction (*Fig. 236*).

Aug. 11.—X-ray shows that reduction has proceeded, and the margin of the head is now continuous with the upper margin of the neck (*Fig. 237*).

Sept. 25.—X-ray shows reduction is now complete, the criteria being: (1) Shenton's line is normal; (2) The epiphysis has assumed its normal elliptical shape instead of the sphere of the earlier films. This film also shows the development of new bone at the lower end of the epiphysial line where the periosteum was stripped off by the original trauma (*Fig. 238*). The patient was now taken off the frame and allowed free active movements in recumbency.

Oct. 3.—The condition noted was: No pain; about 20° active movement present in all directions; Bryant's triangle showed a small amount of real shortening, less than $\frac{1}{2}$ in.; considerable wasting of gluteal muscles.

Oct. 7.—Confirmed reduction (*Fig. 239*).

Oct. 24.—Shortening was still just measurable. Movement had increased to: Abduction, 45°; flexion, 45°; adduction, full; external rotation, full; internal rotation, nil. There was noticeable recovery in gluteal muscles.

Nov. 7.—It was curious that shortening had now disappeared, although traction had been stopped for some time. Abduction, 45°; flexion, 45°; adduction, full; external rotation, full; internal rotation, half range.

Feb. 20, 1932.—Flexion had increased to a right-angle, other movements remained the same. The patient was fitted with a walking caliper and exercises continued as an out-patient.

Sept. 10.—Abduction, full; flexion, 90°; adduction, full; external rotation, full; internal rotation, limited.

January, 1933.—This patient has been actively employed for the last six months and has now applied for admission to the Police Force.

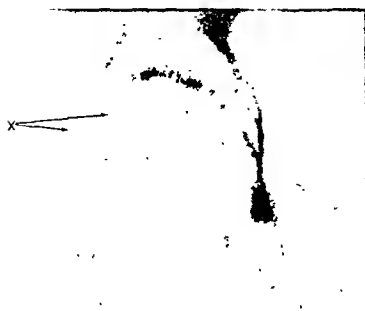


FIG.
235.

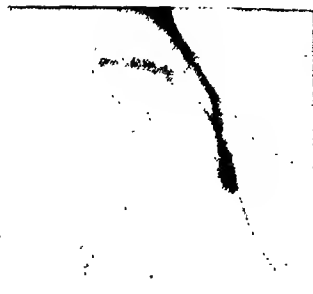


FIG.
236.

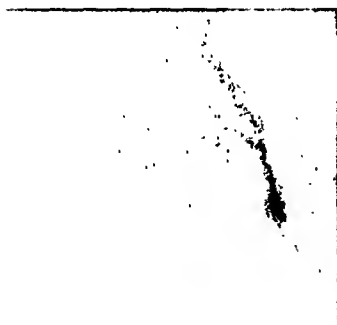


FIG.
237.



FIG.
238.



FIG.
239.

FIGS. 235-239.—*Case 1.*
X-rays showing progress of treatment.
X shows areas of rarefaction.

FIG. 235.—July 2, 1931.

FIG. 236.—July 23, 1931.

FIG. 237.—Aug. 11, 1931.

FIG. 238.—Sept. 15, 1931.

FIG. 239.—Oct. 10, 1931.

FIG.
240.



FIG.
241.



FIG.
242.



FIG.
243.



FIG.
244.



FIG.
245.



FIGS. 240-245. —Case 3. X-rays showing progress of treatment.

X shows areas of rarefaction.

FIG. 240. —Nov. 20, 1931.

FIG. 241. —Dec. 22, 1931.

FIG. 242. —Jan. 23, 1932.

FIG. 243. —Feb. 27, 1932.

FIG. 244. —April 2, 1932.

FIG. 245. —July 19, 1932.

Case 3.—C. R. W. H., male, aged 14 years. One month previously the patient had noticed 'growing pains' in the left hip and had been noticed to limp a little. A week previously he had knocked his left ankle while going upstairs; and this gave rise to pain again; later he found he could not walk. When examined on Nov. 20, 1931, the leg was held everted and abducted at the hip-joint and there was a tense, painful swelling about the size of an egg in Scarpa's triangle. There was 20° active flexion at the hip but all other movements were limited by muscle spasm. Attempted movement caused pain. On measurement there was 1 in. real shortening.

Nov. 20, 1931.—X-ray shows typical slipped epiphysis (*Fig. 240*). The patient was placed on a frame in the manner described. Unlike *Case 1*, all the difficulties which are made so much of by the opponents of fixed traction were encountered, but as will be seen by the notes below they were all successfully overcome without resort to any other method of treatment; furthermore, the patient was never subjected to the risk of an anaesthetic, nor was the hip traumatized by violent manipulation at any time.

Dec. 10.—The commencement of a pressure sore was observed in the left groin and the inversion bandaging around the leg had produced œdema of the foot. The patient was therefore taken off the frame. In order that no ground might be lost, the foot of the bed was raised and the patient suspended by tying the extension tapes to the bed rail; a pillow was placed behind the knees.

Dec. 19.—Real shortening was still 1 in., and the pressure sore was not healed. A weight was attached to the extensions of the left leg to prevent any shortening while off the frame.

Dec. 23.—Control X-ray showed no change in the position (*Fig. 241*). As the sore had now healed, the patient was put back on the frame, but very light traction was used to start with and the strap kept on the right side temporarily.

Jan. 9, 1932.—A right foot-drop developed and the strapping was removed from the area of the external condyle of the femur and the head and neck of the fibula.

Jan. 16.—The groin strap had now been replaced on the side of the lesion and power had returned to the right anterior tibial group of muscles.

Jan. 23.—X-ray showed marked improvement in the position of the epiphysis (*Fig. 242*).

Feb. 20.—X-ray showed the epiphysis almost in its normal position. Measured by Bryant's triangle no shortening was recorded (*Fig. 243*).

March 19.—Removed from frame. Active movements as follows were noted immediately the patient was free: Abduction, 30°; flexion, 25°; little rotation. Active exercises in recumbency were commenced.

April 2.—X-ray confirmed position (*Fig. 244*).

May.—During May the patient was allowed to walk in a caliper.

July 16.—X-ray showed the position to be maintained (*Fig. 245*). Active movements: Abduction, 30°; flexion, 90°; external rotation, full; internal rotation, half range.

January, 1933.—The patient is now walking well without his splint.

Case 11.—A. G. H., male, aged 17 years. This youth was first seen on April 13, 1933. He stated that he had noticed that he was walking with a limp for eight weeks previously, and that his left knee and ankle 'gave way' easily. There was no history of any previous accident or illness.

On examination he was found to lie with the left leg externally rotated at the hip, and there was $\frac{3}{4}$ in. real shortening present. Abduction and internal rotation were limited at the hip-joint and there was 1 in. wasting of the quadriceps $\frac{1}{2}$ in. above the superior margin of the patella. X-ray (*Fig. 246*) shows slipping of the left upper femoral epiphysis. (Compare with X-ray of normal hip, *Fig. 247*.)

Fixed traction, as described earlier, was commenced in a few days, as soon as a frame could be obtained, and a series of X-rays taken during treatment. *Fig. 248* shows the condition after three weeks' traction. The relationship between capital epiphysis and femoral neck has definitely altered. The outline of the head and

SLIPPED EPIPHYSIS OF HEAD OF FEMUR 325

upper margin of the neck are no longer continuous and Shenton's line is nearer to the normal. *Fig. 249* was taken after six weeks' traction. Correction has progressed and Shenton's line is now normal. Skiagrams taken at the end of eight



FIG. 246.—*Case 11.* Condition when diagnosis was made.



FIG. 247.—*Case 11.* Skiagram of normal hip.

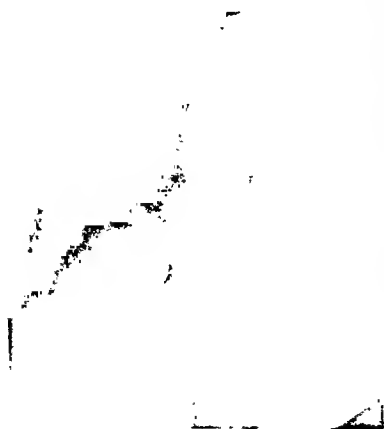


FIG. 248.—*Case 11.* Condition after traction for three weeks.



FIG. 249.—*Case 11.* Final condition on removal of frame.

weeks showed no further correction, and clinical measurement confirmed full length of the limb. The patient was thereupon removed from the frame.

Active non-weight-bearing exercises were then started, in recumbency, and have been continued to the present time. The range of movement at the left hip

is now : Flexion, 90° ; abduction, 45° ; rotation, the foot can be rotated internally as far as the foot of the normal leg. Measurement shows that full-length is maintained, and there is no pain. The patient has now been measured for a walking caliper.

SURVEY OF METHODS AND RESULTS.

Key¹ records eight cases in which either manipulation or operation were used ; 50 per cent of these had shortening of 1 in. or more afterwards. Half of them had a flexion deformity and 45° was the maximum of abduction obtained. He rightly concluded that forcible reduction of the displaced epiphysis by the Whitman method was a fertile source of stiff hips subsequently, and recommended reduction by an open operation in cases where bony consolidation had not occurred.

Badgely² followed up Key's suggestion, but out of six cases reported as operated on, two were of the Whitman reconstruction type, one was an osteotomy, and two of the remaining three developed non-union. Also the standard of comparison was not high, as one case with $\frac{3}{4}$ in. shortening was quoted as a good result.

Willis³ later in the same year recommended manipulation, but gives no definite after-results. Irwin⁵ does not record any after-results. Jahss⁶ records three cases in detail and describes his own modification of the method of reduction by forcible manipulation. His results are good and he shows X-rays confirming the reduction in each case. Taylor¹⁰ quotes a series of Liverpool cases which he had collected. Fourteen of these were old enough to be considered after-results, and included cases which had had no treatment, forcible manipulations, and reduction by open operation. The great majority of the results were poor.

After a consideration of these records the following points suggested themselves :—

The methods in use for the reduction of this deformity, up to the present, are open to serious criticism on the grounds that they do not produce a functionally useful hip, and function is the only real test of success.

Some definite standard of result must be laid down at which the treatment should aim, and I would state it thus : The patient should have a painless hip and a minimum of 90° active flexion and 45° abduction before the result is considered good. Further, there should not be more than $\frac{1}{2}$ in. real shortening.

Far too much emphasis has been laid upon anatomical reduction as shown by X-rays, and the functional recovery of the hip-joint has been lost sight of. Anatomical reduction is worthless if the price of a stiff hip has to be paid for it.

Further, a study of a series of X-rays has convinced me that forcible abduction of the hip in these cases is wrong in principle and practice ; as can be seen from *Fig. 250*, when the femur is abducted the lower edge of the metaphysis engages the separated epiphysial surface and tends to force the epiphysis even farther downwards and backwards in the acetabulum. In effect, this means that the leverage which is being used to effect reduction has its fulcrum at the point where articular cartilage of the head impinges

SLIPPED EPIPHYSIS OF HEAD OF FEMUR 327

on the lower and posterior acetabular margin; the result is bruising of the cartilage and a subsequent arthritis of the traumatic type; and even when reduction starts, the metaphysial edge ploughs through the cervical surface of the epiphysis. Together with the tearing of capsule and ligamentum teres, mentioned by other authors, the damage is quite sufficient to account for subsequent stiffness.

Considered from the anatomical point of view alone, the close fit of the head of the femur in the acetabulum makes it imperative that the reducing force should be in the long axis of the femur and not in a direction which forces the head against the acetabular wall.

The paradox that "untreated cases of slipped epiphysis do better than treated cases", stated by Key¹, is capable of a simple explanation on these lines; in striving after perfect anatomical reduction, function has been quite forgotten. A similar conclusion is reached by Perkins⁷ in his article.

The idea of traction applied gradually, without abduction of the hip, has been evolved in response to the demands laid down earlier, and eliminates any possibility of damage to articular cartilage or capsule and blood-vessels, thus leaving the way clear for the future development of a movable and useful joint. At the same time the method holds out the prospect of at least as good a reduction as claimed for other methods, although this is not made the criterion.

I do not agree with Perkins⁷ that gradual traction should be confined to any particular type of case. It is worth a trial in all cases and a period of seven days is not long enough in which to judge of its efficacy. It is agreed that for the old case, consolidated in deformity, subtrochanteric osteotomy is excellent treatment, and Case 10 of my series bears this out, but it is surely wrong to fall back deliberately on to a second line of defence when simple traction is likely to succeed if persisted with.

Open operation is condemned by the statistics of those who have tried it.

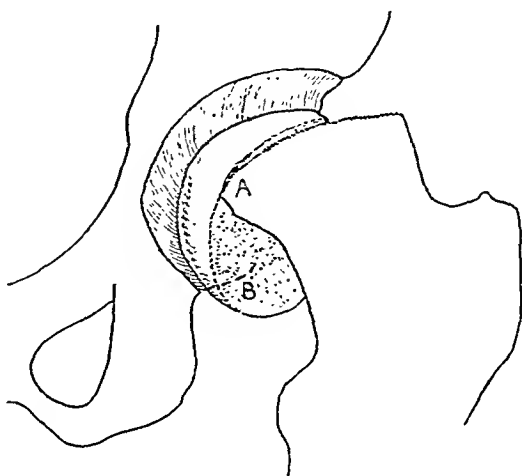


FIG. 250.—Line drawing from skiagram of recent case of slipped epiphysis. A shows point where lower edge of metaphysis engages separated epiphysal surface; B indicates fulcrum of leverage force when femur is abducted.

SUMMARY.

1. The etiology of slipped epiphysis is considered, and it is suggested that either indirect trauma or endocrine disorder produces weakening at the epiphysal line: displacement then proceeds under the force of body weight.

2. Treatment by gradual traction in adduction is described and three successful cases are reported in detail with serial X-rays.

3. Gradual traction is advocated as being the most efficient form of treatment. Subtrochanteric osteotomy is an excellent second line of defence, but should be reserved for cases seen when the deformity has consolidated. No case should be allowed to consolidate in deformity without a prolonged trial of traction. Open operation is condemned.

I wish to thank Mr. T. P. McMurray for his guidance in the treatment of *Cases 1 and 3* at Alder Hey Hospital; Mr. T. R. W. Armour for permission to publish *Case 8* from the Royal Southern Hospital, Liverpool; Mr. W. J. Eastwood for facilities to examine *Cases 2 and 4* at the Wigan Infirmary; Mr. C. O. Davies for *Case 5* from Liverpool Stanley Hospital; and Mr. C. A. Wells for *Case 7* from the Clatterbridge Institution, Cheshire. Mr. V. J. M. Taylor very kindly allowed me access to a series of earlier cases which he had collected in Liverpool, and which he has published recently.

BIBLIOGRAPHY.

- ¹ KEY, *Jour. Bone and Joint Surg.*, 1926, xxiv, 95.
- ² BADGELEY, *Jour. Amer. Med. Assoc.*, 1929, xcii, 355.
- ³ WILLIS, *Jour. Bone and Joint Surg.*, 1929, xxvii, 779.
- ⁴ ELSLIE, *Lancet*, 1907, Feb. 16.
- ⁵ IRWIN, *Irish Jour. of Med. Sci.*, 1929, Feb., 71.
- ⁶ JAESS, *Jour. Bone and Joint Surg.*, 1931, xxix, 856.
- ⁷ PERKINS, *Brit. Med. Jour.*, 1932, Jan. 9.
- ⁸ LERICHE and POLICHARD, *Pathological Physiology of Bone*.
- ⁹ KOCHER, *Deut. Zeits. f. Chir.*, 1893-4, xxxviii, 521; 1894-5, xl, 411.
- ¹⁰ TAYLOR, *Brit. Med. Jour.*, 1932, ii, 1003.
- ¹¹ DE QUERVAIN, *Clinical Surgical Diagnosis*, 1926, 778. London.
- ¹² KIRMISSON, *Différentiels acquises de l'Appareil locomoteur*, 1902, 379. Paris.

OBSERVATIONS UPON MULTIPLE INTRAMESENTERIC DIVERTICULA OF THE SMALL INTESTINE.

By R. WEEDEN BUTLER,

SURGEON, ADDENBROOKE'S HOSPITAL, CAMBRIDGE.

THE condition of multiple intramesenteric diverticula of the small intestine is a definite pathological state, which must be distinguished from others in which diverticula occur in the small bowel at its mesenteric border, such as traction diverticula secondary to adhesions or to the scarring of old inflammatory lesions, congenital pouches associated with accessory gland tissue, and vestigial and developmental cysts and diverticula.

Published records, both in this country and abroad, are rather scarce, and many are insufficiently thorough in details of the patient's past history and symptoms and in the cause of death to be of any assistance in studying the subject as a whole, consisting only of a bare description of the naked-eye pathology.

This paper is based upon a detailed study of seven new cases collected by the writer. Three of these were observed personally as patients in the wards of St. Thomas's Hospital, and in two of these and in the four others material was available for pathological investigation.

For the purpose of a more comprehensive survey these seven have been considered together with thirteen others previously recorded with sufficient detail, both clinical and pathological, to assist in an investigation of the origin and significance of the condition. Of these thirteen, twelve have been described in various medical journals. These are listed under the names of the authors in the reference table at the end of the paper.^{1-5, 8, 10, 11, 13-15} The thirteenth case is that from which was obtained specimen No. 3065 K.L. in the Museum of the Medical College, University College Hospital, where the case notes are also preserved. These thirteen, with the writer's seven, make a total of twenty fully recorded cases which have been used as a source of general and statistical information in compiling this paper.

In the present number of this JOURNAL another writer (Ian Fraser: "The Diverticula of the Jejunum-ileum") has made a general survey of the whole subject (p. 183). It is therefore unnecessary to repeat a great deal that is already written, and in this paper the more general features of the condition will be described briefly first, and the writer will confine himself to a full description only of his own cases and of the conclusions reached by clinical and pathological study.

GENERAL CONSIDERATIONS.

The condition is rare. Often it has been found accidentally at the post-mortem examination of persons who have died from some totally different cause. Nineteen out of twenty mentioned by Terry and Mugler¹⁵ were so

found. In such there is frequently no history of symptoms attributable to the condition of the bowel, and we are therefore led to wonder whether, as J. T. Case⁵ suggests, the condition (though rare) may not be somewhat more common in elderly persons than is usually supposed.

In the twenty cases upon which this paper is based the average age at which the condition was discovered is 64, and the writer can find no description of multiple diverticula of this type being found in a child or young person. In some of these cases the diverticula were no doubt of long standing, but in most of the younger cases the diverticula were few in number, and it is evident that the condition is developed typically in persons past middle age, and probably seldom becomes apparent before the age of 40 at least.

The twenty cases in this series comprise fourteen men and six women.

THE GROSS ANATOMY OF THE CONDITION.

Once seen, this condition can never be mistaken, so remarkable is the appearance when well developed (*Figs. 251, 252*). A number of diverticula are seen in the jejunum and upper part of the ileum. They are always most plentiful in the first two or three feet beyond the duodenojejunal junction, and when only two or three are present they are found in the first jejunal loop. When a very large number occur—as many as four hundred have been recorded—they sometimes extend for several feet along the bowel, and at times are scattered throughout almost the whole length of the ileum as well as the jejunum. In such, however, the larger diverticula lie close to the duodenojejunal junction.

In the same specimen the diverticula may vary in size from small conical poekets the size of a pea to large globular diverticula 7 cm. or more in diameter (*see Figs. 251, 252*). All except the very small sacs have obviously thin walls, though here and there the larger sacs may be thickened by fine adhesions to the adjacent bowel.

The diverticula all occur at or near the mesenteric border. The small ones push straight into the mesentery, but as they increase in size they bulge out from the side of the mesentery, usually towards the right. Finally, the very large diverticula tend to stand away from the mesentery and to overlap the bowel, completely covered by a peritoneal coat except at their broad base of attachment.

Klebs¹² in 1867 was the first writer to point out that there is a definite relationship between the mesenteric blood-vessels and the diverticula. Each diverticulum arises from a point at which one of the vasa recta of the superior mesenteric artery reaches the bowel wall.

In the case of the small diverticula this is readily made out (*see Fig. 252*), but with the very large diverticula this relationship to the vessels is lost, as the latter are pushed to one side by the enlarging sacs. The exact significance of the vessels in the mode of origin of the diverticula will be discussed later. An interesting feature is that there are usually more diverticula to the right of the mesentery than to the left. Probably this is because there is more room for them towards the right, since the first loop of the jejunum lies over towards the left and its mesentery is in contact on the left with the rigid

MULTIPLE INTRAMESENTERIC DIVERTICULA 331

structures forming the posterior wall of the peritoneal cavity. Where the diverticula appear for more than a couple of feet down the jejunum this partiality of the diverticula for the right side of the mesentery disappears, which fits in well with this explanation.

The orifices of communication between the bowel and the diverticula are large. The smaller diverticula, 1 cm. across, have orifices opening into the bowel almost as wide as their own greatest diameter. A larger diverticulum, 2.5 cm. in diameter, will have an orifice perhaps 1.5 cm. wide. The largest diverticula, 6 or 7 cm. in diameter, have orifices 2 to 3 cm. wide.

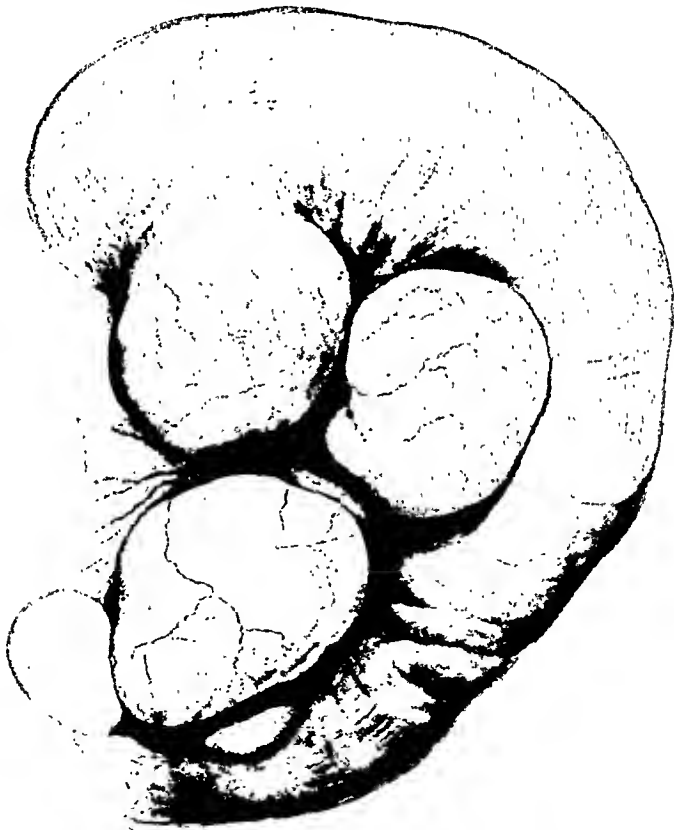


FIG. 251.—Sketch of part of specimen from a case described by Mr. L. R. Braithwaite. Shows the great size to which diverticula of this type can grow, these being easily the largest that the writer has seen in any specimen of this condition. Small commencing diverticula are also seen. ($\times \frac{1}{4}$)
(Specimen in Museum of the Royal College of Surgeons.)

THE CLINICAL STATE.

Clinically, patients fall into three groups:—

1. Those in whom the condition is discovered purely by accident—perhaps post mortem—and in whom the state of the bowel has produced no symptoms during life and is in no way directly related to the cause of death.

2. Those in whom there are definite symptoms produced by the diverticula during life.

3. Those in whom symptoms, if they have been present at all, have been comparatively slight, but who are seen because of an urgent abdominal emergency directly related to the presence of the diverticula.

Twelve of these twenty cases fall into Class 1 in that there is no history of any symptoms definitely attributable to the condition previous to its discovery. The first of the writer's new cases is an example of this type.

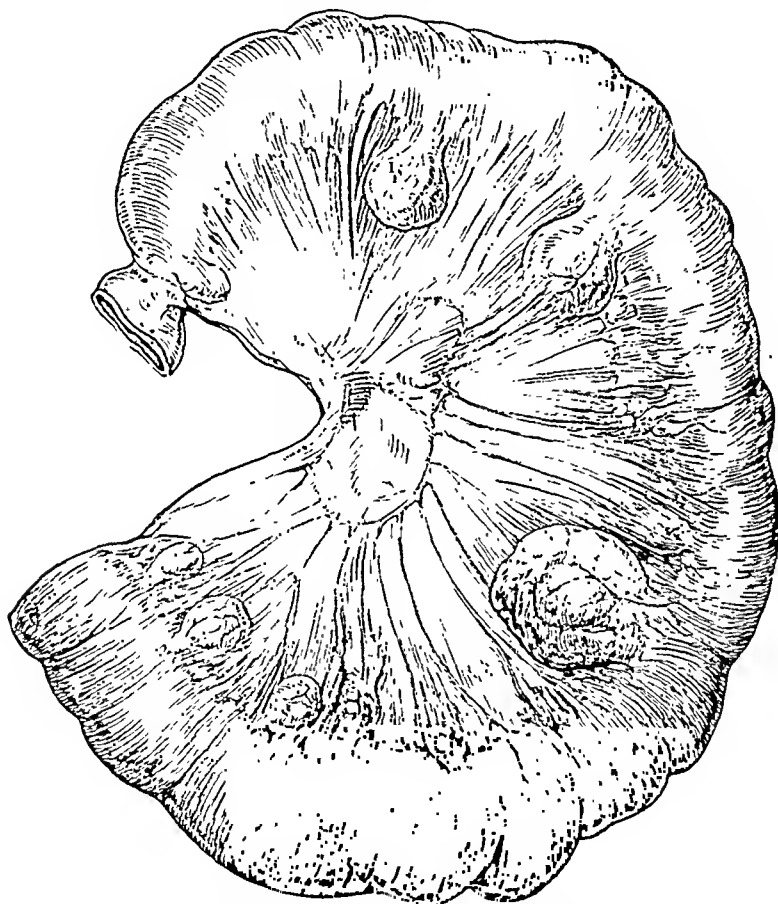


FIG. 252.—*Case 1.* The first 16 in. of the jejunum seen from the right. Diverticula of many different sizes are seen, and the direct relationship of the vasa recta to the points at which they arise is in some cases clearly evident.

Case 1.—A. S., female, aged 65. Admitted to Lewisham Hospital in January, 1926, suffering from a vaginal recurrence of carcinoma of the body of the uterus, for which a hysterectomy had been previously performed elsewhere. A bleeding mass was present in the vaginal scar. Within a few days this patient developed subacute intestinal obstruction of large-bowel type. General peritonitis rapidly supervened and the patient died.

AUTOPSY.—At the post-mortem examination the cause of the obstruction was

found to be a carcinoma of the sigmoid colon. A stereoral ulcer of the cæcum had perforated. There were a number of diverticula in the sigmoid colon.

In the upper 2 ft. of the jejunum were some twenty diverticula at the mesenteric border, varying in size from a small pea to a large walnut. *Fig. 252* shows the specimen of the jejunum from this case. The relationship of the vessels to the diverticula is well shown. The histology of this specimen will be described later.



FIG. 253.—*Case 2.* X-ray six hours after bismuth meal, showing multiple fluid levels in the diverticula, many with gas above. The diverticula in this case were many of them large and they were distributed throughout the whole of the jejunum. Probably hundreds were present.

During life these diverticula were quite unsuspected, and any dyspeptic symptoms that may have been present were not sufficiently severe to attract attention.

(For the clinical details the writer is indebted to Dr. H. Noekolds, Medical Superintendent of Lewisham Hospital.)

On the other hand, eight cases in the series of twenty show certain chronic symptoms attributable to the abnormal condition of the bowel during life. The writer's second case is of this clinical type.

Case 2.—H. L., male, aged 63. Admitted to St. Thomas's Hospital under the care of Dr. Cassidy. 'Two years' history of abdominal discomfort and wasting. This began as slight constant flatulence; always feeling as if he wanted to "bring up wind", with a certain amount of mild aching pain in the abdomen. As time went on the flatulence became worse, and he began to vomit, at first only occasionally, but much more frequently for the last six months. The more frequent vomiting was often preceded by acute attacks of pain across the upper part of the abdomen. Of late the vomit had contained a good deal of undigested food, and had occurred at all hours of the day and night. Vomiting did not now relieve the pain completely. For the last six months particularly he had been losing weight rapidly. For several months he had also been very constipated.

ON EXAMINATION.—Very wasted; edentulous. Abdomen diffusely tender and showing an indefinite fullness in the upper part towards the right. This fullness was somewhat hyper-resonant, suggesting dilated bowel. General arteriosclerosis very marked. Pulse 80, blood-pressure 120/90. An X-ray following a bismuth meal showed a very remarkable appearance of multiple fluid levels throughout the small intestine (*Fig. 253*).

OPERATION.—A laparotomy was performed, revealing the following state of affairs: The small bowel generally was slightly dilated, especially in the upper part. A great many, probably hundreds, of diverticula were present in the small bowel at the mesenteric border. They varied in size from a pea to a tangerine orange. The larger diverticula were in the upper part of the small bowel, and they gradually lessened in their average size as the bowel was examined downwards. The diverticula contained mainly gas. No short-circuiting operation that would be likely to benefit the patient was possible, and he was considered unfit for an excision of the whole of the affected bowel. The abdomen was therefore closed.

SUBSEQUENT PROGRESS.—Healing was rapid, and with rest in bed, small and frequent meals, strict attention to the bowels, and the taking of small doses of carminatives to keep the stomach free from distension with gas, the patient gradually became much more comfortable. Alkalis in the form of bismuth carbonate helped to relieve his symptoms. About a month after the operation, however, the patient began to show signs of cerebral degeneration of a senile type. This man was, however, still alive in comparative comfort six months later.

Another example of rather similar but less marked symptoms is shown in the following—the third of the writer's cases.

Case 3.—W. W., male, aged 61. Had complained for "some months" of flatulence, vague abdominal pain, and a feeling of fullness after meals, but no real pain. There had been no vomiting. He had been rather constipated for six months. He had actually been increasing in weight and getting stouter for several months. Two months before his death he went to his doctor complaining of these vague symptoms, but the only discoverable signs were very marked general arteriosclerosis and a pronounced degree of myocardial degeneration. One day, while leaving a train at Waterloo Station, he suddenly fell dead.

AUTOPSY.—At the post-mortem examination (held at St. Thomas's Hospital) there was found marked general arteriosclerosis, with atheroma of the aorta and much narrowing of the coronary vessels. The first 4 ft. of the jejunum showed about fifty diverticula.

J. T. Case⁵ in one of his patients found a history, very similar to the above, of mild 'indigestion' for many months; a feeling of pressure and distension following meals, relieved somewhat by belching. No nausea or vomiting. No real pain, but rather abdominal discomfort. For six months there had been much intestinal flatulence.

DIAGNOSIS.

The abdominal symptoms are not distinctive, and in the absence of any X-ray evidence of the diverticula it is certain that a diagnosis of some much more common condition would be made. In *Case 2* the symptoms suggested simply a gradually increasing degree of small-bowel obstruction. In such cases X-rays following an opaque meal would seem to be the only way of making a certain diagnosis other than by laparotomy.

X-ray Diagnosis.—J. T. Case claims to have diagnosed five cases in this way—two of them checked by laparotomy. Gas accumulation in the diverticula was seen on the screen before giving the bismuth meal in some of his cases.

Fig. 253 shows very strikingly indeed the multiple fluid levels with gas above them six hours after a bismuth meal, and shows the condition in *Case 2* described above. The diverticula were seen to be almost clear of bismuth ten hours after the meal, and the small bowel appeared to empty within the normal time limit. This bears out Case's statement that the diverticula take eight to ten hours to empty, and checks the supposition which we have already made on anatomical grounds that the wide openings of the sacs would not retain the fluid bowel contents for long. Peristaltic movements of the bowel pressing on the thin-walled diverticula would doubtless empty them easily of their fluid contents, and it is perhaps an accumulation of gas, rather than of fluid bowel contents, which gives rise to the feeling of fullness of which the patient complains.

Having once seen the typical X-ray appearance of fluid levels in the diverticula with gas accumulation above as in *Fig. 253*, it would be imagined that the diagnosis would easily be made by X-rays whenever the diverticula were large and numerous. Curiously enough this is not always so. In *Case 7*, to be described later, repeated X-rays taken after recovery from the operation for peritonitis failed to show clear evidence of any diverticula, though more than a dozen large sacs were known still to be present. Other writers have noted this in individual patients, and it is probable that the amount of retention in the diverticula is much greater at some times than at others, varying perhaps with the tone of the involuntary muscle of the bowel wall.

TREATMENT OF THE UNCOMPLICATED CONDITION.

If found accidentally by X-rays in a person in whom it is producing no symptoms, the condition should be left alone without surgical interference. If troublesome symptoms are present, an exploratory operation is certainly justifiable, as in some cases, such as that described by L. R. Braithwaite,³ a complete resection of the affected area of bowel may be possible, and this is undoubtedly the most satisfactory treatment where it is practicable, for, as we shall see, acute complications of the condition may arise without warning. Should the diverticula be too widely scattered for this, a short-circuit operation consisting of an anastomosis of the upper and lower ends of the affected area may relieve the symptoms to some extent and serve as a prelude to a later excision when the patient shall be in a better state to stand the more severe operation.

Inversion or excision of individual diverticula, as Gordinier and Sampson⁸ point out, is likely to endanger the blood-supply of the bowel wall locally, and is obviously impracticable as a radical treatment where there are a great number of diverticula.

The treatment if acute complications should arise will be discussed later.

If symptoms have only recently begun to appear, it is worth while trying a regulation of the diet, so that meals are small in bulk.

The bowels should be regulated and kept freely open. In the cases which the writer himself has seen, alkalis in the form of sodium bicarbonate and bismuth oxy-carbonate in a simple mixture give relief to the discomfort. Carminatives of any sort seem to help; and, theoretically, tonics and stimulants to the muscular action of the bowel wall should be of value. Rest in bed alone appears to help some patients, and indeed simply lying down for an hour often relieves completely a severe attack of discomfort.

ACUTE COMPLICATIONS.

The gross anatomical abnormality present in many of these cases might lead to the expectation not only of the production of chronic symptoms, but also the sudden advent of acute complications. We have seen, however, that in many there are no chronic symptoms at all, and in the same way sudden disasters due to the presence of these diverticula appear to be rare.

The large size of the openings of the diverticula into the bowel is probably the significant feature in this freedom from complications, for there is very little retention of the bowel contents, which are fluid, and, incidentally, comparatively sterile, in this part of the intestine, so that no inflammation is set up in the walls of the diverticula. When an inflammation does arise, it seems to start in the loose connective tissue lying in the mesentery around the neck of a diverticulum. There is often a considerable bulk of loose fatty tissue lying here, especially immediately above and below the neck of the sac where the peritoneum bridges across the corner between the bowel and the diverticulum.

Here, too, the peritoneum is often thickened by many fine adhesions across from the sac to the bowel. These intramesenteric spaces, often of considerable size and filled with loose areolar tissue and fat, are separated from the lumina of the larger diverticula only by thinned mucous membrane. In Gordinier and Sampson's case this region had been the site of an inflammation which had obliterated the neck of a diverticulum. In three of the four new cases about to be described infection with perforation into the peritoneum had occurred at this point. In the fourth the site of the commencing inflammation could not be identified.

The writer's four cases of acute inflammation arising in this condition are as follows:—

Case 4.—Female, aged 72 (February, 1923).

HISTORY.—Occasionally constipated, but bowels usually regular. Awoke one morning at 5 a.m. with sudden acute pain in abdomen, localized to the left side just above the umbilicus. Vomited once; abdomen rigid, slightly distended. Twelve hours later pain was less severe and vomiting had diminished.

ON EXAMINATION.—Pulse 120, temperature subnormal. Tongue dry and brown. Abdomen motionless and rigid. Acute tenderness to left of umbilicus. No loss of liver dullness. Diagnosis: perforation of some hollow viscus.

OPERATION.—Laparotomy. Small quantity of turbid fluid escaped on opening the abdomen. On left side, deep to tender area, was a perforated diverticulum in the mesenteric attachment of the upper part of the small intestine. In the adjacent part of the bowel on either side were several more diverticula, all at the mesenteric attachment. Two feet of the intestine were resected, and continuity restored by axial anastomosis. Adjacent portions of the intestine showed further diverticula. Recovery was complete and uneventful.

The perforation had occurred at the junction of the sac and the bowel wall, where a suppurative inflammation extended from the peritoneum as far as the mucous membrane, but the mucous membrane itself around the perforation showed but slight signs of acute inflammation. The wall of the sac consisted of fibrous tissue, containing only traces of muscle between the peritoneal and mucous coats. In the bowel wall the muscle was normally developed. (Described by permission of Mr. C. R. Nitch.)

Case 5.—Male, aged 56 (Aug. 24, 1928).

HISTORY.—Was accustomed for many years to suffer from "bilious attacks" with abdominal pain, vomiting, and diarrhoea, recurrent almost weekly, but these had ceased for twelve years. No indigestion since. Twelve hours before admission to hospital sudden epigastric pain stopped him working; he belched up wind at once. Three hours later he began to vomit. Pain was worse on taking a deep breath.

ON EXAMINATION.—Pulse 70, temperature 98°, respiration 20. Tongue furred and dry. Tender and rigid between costal margin and umbilicus, whole belly rigid. Liver dullness much diminished.

OPERATION.—Laparotomy. Gas and blood-stained fluid in peritoneum. Stomach and duodenum normal. About 1 ft. of the upper jejunum found to be hyperæmic, with hæmorrhages into the peritoneum and mesentery. Five large diverticula at mesenteric border; one of these had perforated. All this portion of gut was resected, and end-to-end anastomosis was carried out.

In the course of the next three days paralytic ileus developed and failed to respond to all palliative treatment. A jejunostomy was performed under local anaesthesia, the tube being passed into the bowel below the line of anastomosis and up the lumen through the anastomosis. The tube drained well, but the patient died on Aug. 28. (Clinical details supplied by Mr. D. C. Taylor, Lewisham Hospital, and specimen loaned by Dr. Barnard, late Curator of Medical College Museum, University College.)

Case 6.—Female, aged 72 (March 4, 1928).

HISTORY.—Always very healthy. No history of any sort of abdominal trouble. A wiry old lady. Thirty-six hours before admission she suddenly had a severe attack of vomiting. Pain in left side of abdomen, high up at first, and later generalized all over. Could keep no food down. Bowels open twice.

ON EXAMINATION.—Abdomen distended. There was general tenderness and rigidity. Pulse 100, temperature 98°. Nothing abnormal was felt per rectum.

OPERATION.—Laparotomy. Free turbid fluid in peritoneum. Appendix normal. In the left iliac fossa close to the sigmoid colon was a coil of small bowel bent round and adherent to itself. On straightening out this coil a small abscess was found in the convexity of the loop about a perforation in the neck of a diverticulum. This was one of seven diverticula in the upper 2 ft. of the jejunum, all running into the mesentery from the mesenteric border of the bowel. The mesentery was very oedematous and injected. About 9 in. of the jejunum were resected and end-to-end anastomosis obtained (Fig. 254).

For two days after operation the patient's condition was satisfactory, but she then began to sink gradually, becoming slowly more and more comatose.

Finally suppression of urine took place and she died with symptoms of uræmia on March 10.

Microscopy showed acute inflammation of the whole thickness of the wall of the diverticulum at the junction with the bowel.

AUTOPSY.—At the post-mortem examination five more diverticula were found in the jejunum, one being at the duodenojejunal junction, and the four others about 2 ft. below. (Surgical Unit, St. Thomas's Hospital.)

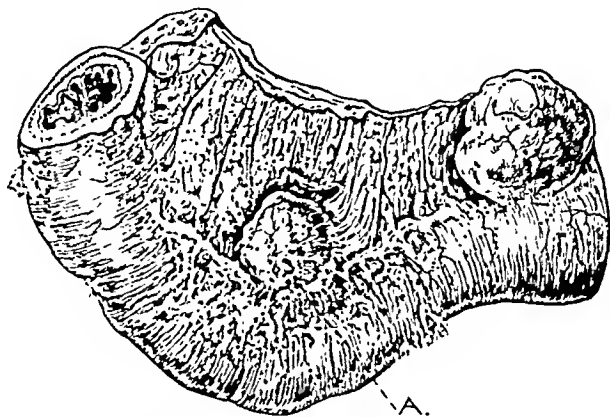


FIG. 254.—Case 6. Part of a loop of jejunum resected at operation for acute perforation of a diverticulum. The loop was found bent round and adherent to itself over the site of the perforation (A). Note the great œdema of the mesentery and the adjacent bowel wall, and the adherent fibrinous conglutination.

Case 7.—Male, aged 64 (March 5, 1928).

HISTORY.—Quite blameless; no abdominal trouble of any sort. A lean, wiry man. Sudden epigastric pain the day before admission, moving from side to side. Vomited several times.

ON EXAMINATION.—Abdomen distended and rigid. Pain continuous all over abdomen, worse in mid-line and umbilical region. Breathing laboured. Tenderness worse on left side. Nothing abnormal was felt per rectum.

OPERATION.—Laparotomy. Glairy pus free in the peritoneum. Jejunum inflamed, and of a bright pink colour for first 18 in., gradually fading off below. Both the bowel and the mesentery in this region were extremely œdematous. There were a great number of diverticula varying in size from a pea to a pigeon's egg, projecting into the mesentery throughout the inflamed area, and for several feet beyond. An indefinite mass was felt low down in the wall of the sigmoid colon, thought at the time to be an area of diverticulitis as it was not hard enough for a carcinoma. As there was no definite point of perforation visible, and as the patient's condition did not warrant any attempt at resection, the peritoneum was mopped out and the abdomen closed. Some suppuration occurred in the rectus sheath, but otherwise recovery was rapid and uneventful.

Subsequent examination with the sigmoidoscope and barium enemata failed to demonstrate any lesion in the sigmoid colon. A barium meal demonstrated the diverticula in the upper part of the small intestine. Since this operation the patient has suffered from a certain amount of flatulent dyspepsia, but has otherwise been in excellent health. (Surgical Unit, St. Thomas's Hospital.)

It is curious to note that as a remarkable coincidence these last two patients, both with the same rare complication of a rare condition, were admitted for urgent treatment to St. Thomas's Hospital on two consecutive days.

Discussion.—Unless the diverticula are already known to be present, diagnosis of an acute inflammation or perforation is clearly almost impossible. In these cases the preponderance of pain and rigidity on the left side is easily explained, but there are other more likely conditions that could produce this sign, and if the symptoms of the acute complication are the first produced by the condition, diagnosis will only be made at the operation. One of these cases manifested the symptoms of a subacute obstruction, another those of a perforated viscus, and the others those of a spreading peritonitis, but in none of them was there any doubt of the urgent necessity for operation.

As regards the actual procedure at operation, every case would have to be judged on its own merits. In four of the above cases resection was carried out; two recovered and two died. It is possible that the two last would have done better if the peritoneum had simply been mopped out and the area of the perforation buried by sewing over the surrounding tissue. If at the time of operation the condition of the patient is very critical, that would doubtless be the more hopeful procedure, for even under the best conditions a resection of a portion of the jejunum is not an operation that can be depended upon to give consistently happy results.

MINUTE ANATOMY.

The writer has had the opportunity of subjecting to microscopy tissue from four patients. In each case examination was made of sections of the bowel wall, of diverticula of various sizes, of the mesenteric vessels, and of any other available tissue that might be of interest.

The bowel wall between and opposite to the diverticula shows no apparent abnormality. Helvestine¹¹ has reported a reduction in the amount of the circular muscle of the bowel, even in parts of the wall not closely associated with the diverticula, but no evidence of this was found.

In almost every description of the microscopical examination of the walls of the diverticula it is stated that the muscle coats of the bowel are absent, so that the diverticulum consists only of mucous membrane and peritoneum separated by a little connective tissue and fat. The point is an important one. If this is true, it suggests that the diverticula are from the start herniations of the mucous membrane through apertures in the muscle, and any question of a traction effect from without by the vessels or by any other means is at once dismissed.

Evidently the descriptions are largely based on examinations only of the larger diverticula. In these admittedly muscle cells are few, though some evidence of the muscularis mucosæ persists in all but the very largest diverticula. On the other hand, if sections are made of small commencing diverticula, both longitudinal and circular muscle coats are found to be present and continuous, though admittedly somewhat thinned as if by stretching (*Figs. 255, 256*). This was the case in every commencing diverticulum examined by the writer.

In the larger diverticula—especially towards their apices—muscle cells are few and far between. Evidently when the diverticulum reaches a certain

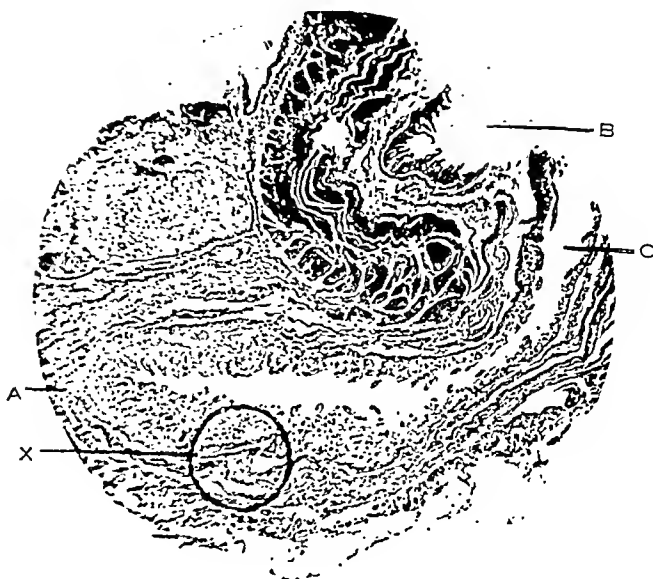


FIG. 255.—Case 1. Section of a small diverticulum 1.5 cm. in length cut in continuity with the adjacent bowel wall and showing the continuity of its lumen with that of the bowel. A, Apex of diverticulum; B, Lumen of bowel; C, Lumen of entrance of diverticulum; X, Area shown further enlarged in Fig. 256. The diverticulum is lying between the layers of the mesentery. ($\times 8$.)

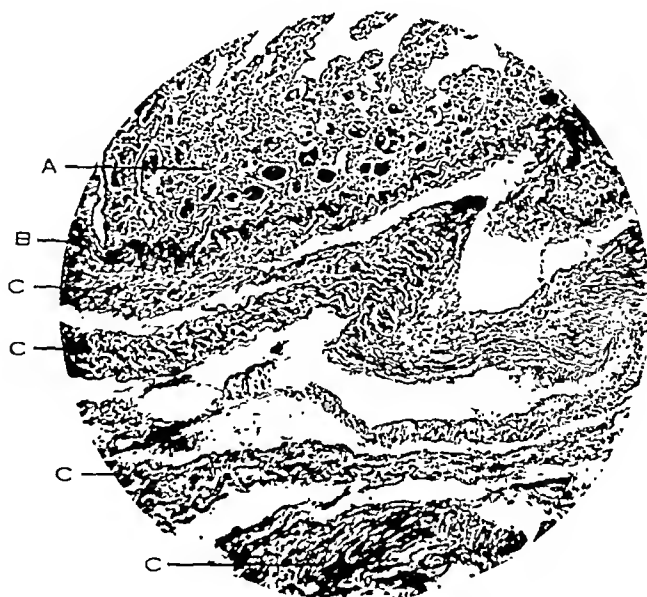


FIG. 256.—Case 1. Part of the wall of the diverticulum shown in Fig. 255. It will be seen that the various layers of the bowel wall are all well represented. A, Villi and glands; B, Muscularis mucosae; C, Muscular wall. It will be seen that the muscular wall is well represented, but the layers are irregular and thinned in comparison with the muscular wall of the bowel. ($\times 50$.)

size—apparently about 1 cm. in diameter—the mucous membrane with the muscularis mucosæ herniates through between the muscle fibres of the main muscular coats. The important fact, however, remains that at the start the diverticula consist of all coats of the bowel. The significance of this in any consideration of the origin of the condition is discussed later.

The mucous membrane as it passes towards the apex of a diverticulum becomes thinner, as if from stretching, and the villi are fewer, shorter, and flattened. These changes are more marked in direct proportion to the size of the diverticulum.

In *Case 5* a section of the wall of a diverticulum close to the area of perforation which had set up peritonitis shows hyperæmia and leucocytic infiltration, as might be expected, but this was the only evidence found of any inflammatory change in the specimens examined.



FIG. 257.—*Case 1*. Transverse section of one of the vasa recta running to the bowel wall opposite a large diverticulum. An area of very great subintimal thickening is seen at X.

Sections of mesenteric lymph-glands from *Cases 3* and *6* show no abnormality.

Transverse sections were cut of the mesenteric vessels in each case, and particularly of the vasa recta approaching the bowel wall opposite the origins of diverticula.

In *Case 1* subintimal thickening and narrowing of the lumen of the vessels is very marked in certain areas, though this is patchy, and other areas appear to have escaped (*Fig. 257*). *Case 3* also shows areas of subintimal thickening and sclerosis of the walls.

Case 6 shows moderate sclerosis, but no marked areas of subintimal thickening in the sections examined.

In *Case 5* only a very small amount of material for examination of the vessels could be obtained, as the mesentery had been cut close to the bowel wall, but the sections examined show no definite evidence of abnormality of the vessels.

ETIOLOGY.

In discussing the anatomy and the clinical manifestations of these diverticula, the following points have emerged and are important in any consideration of their causation: (1) These diverticula are not recorded in infants or young people; (2) They occur rather in persons elderly, or at least past middle age, and often present the appearance of being in the process of progressive formation; (3) Their points of origin at the mesenteric border of the bowel bear a very definite relationship to the vasa recta of the mesenteric vessels; (4) When they are small they consist definitely of all coats of the bowel wall, being true diverticula of the bowel wall, not hernial protrusions of the mucous membrane through the muscular coat.

That these diverticula could be a congenital abnormality, present from an early stage, is denied by the first two of the above statements. Moreover, they show no resemblance to congenital enterogenous cysts and diverticula. Such cysts contain all the elements of the bowel wall, and these are best explained as examples of heterotopia, or an isolation of islands of developing bowel wall at an early stage. A summary of several cases of the type is given by Arthur Evans,⁷ but this writer would appear to include in this category some which resemble rather the particular type of multiple diverticula described in this paper.

Diverticula of the small intestine are occasionally associated with misplaced islands of pancreatic tissue.¹⁶ No evidence of islands of pancreatic or other aberrant gland tissue can be found in the type we have under consideration. There is in fact no evidence that a congenital abnormality plays any part in the causation of this condition. Likewise a weakening of the bowel wall at the mesenteric border by an inflammatory or degenerative process seems out of the question as an underlying cause. In none of the writer's or other reported cases has there been any sign of inflammation of the bowel wall, apart from that associated with acute inflammation or perforation of a well-formed diverticulum.

Another theory of causation—that the mucous membrane herniates out along the sides of vessels passing through the bowel wall, as suggested by Grazer⁹—is denied by two facts. First, the diverticula at their commencement consist, as has been shown, of all coats of the bowel. Secondly, the only vessels with which they are in direct relationship at the start are the vasa recta of the superior mesenteric artery. These, as described by Cockinnis,⁶ run alternately on to one or other side of the bowel, giving off branches at intervals, the first of which runs to the mesenteric border. The main trunk of the vessel does not penetrate the muscle coats at the mesenteric border, so that if penetration were necessary for the formation of the diverticula, these would be as likely to appear at the sides of the bowel as at the mesenteric border.

There remain two important possibilities: the presence of traction upon the bowel from without, and the presence of an increased intra-intestinal pressure acting from within.

Let us first consider the possibility of traction. The definite association of the diverticula with the points at which the vasa recta of the mesenteric arteries reach the bowel wall at once suggests that these vessels might, under certain conditions, exercise a pull upon the bowel wall at these points. Moreover, these diverticula appear first and are more marked in the first loop of the jejunum, the region where the vasa recta are longest and most fixed at their origins.

With a view to ascertaining the effect upon the bowel wall of exerting traction through the mesenteric arteries certain experimental work was carried out by the writer. Fresh post-mortem-room material was used, and in each case the first 12 in. of the presumably normal jejunum was taken and distended tensely with plaster-of-Paris cream. The arteries of the mesentery were also injected with red lead and plaster-of-Paris cream through the superior mesenteric artery.

As the bowel was distended the loop tended to straighten out, and when tensely distended the mesentery in the concavity of the loop formed a diaphragm pulled taut all round its circumference. The vasa recta of the superior mesenteric artery were therefore all put on the stretch. An X-ray of the specimen under these conditions shows the appearance of tiny diverticula opposite the point at which many of the vasa recta reach the bowel wall. The tiny diverticula consisted of all coats of the bowel wall and were therefore clearly small traction pockets. Further distension of the bowel resulted in the rupture of the muscle coats of these small pockets and herniation of mucous membrane, and in one case in bursting of the pocket with escape of plaster cream into the mesentery. An X-ray of a jejunal loop thus distended with plaster cream and with its mesenteric vessels injected, demonstrating these small traction pouches, is shown in *Fig. 258*.

Twelve experimental specimens were thus made, and it is perhaps of interest that the only two in which no pouches appeared were from individuals under 20 years of age, in whom the mesenteric vessels were probably more elastic than in the older cases. Moreover, the traction pouches were best marked in a specimen from a man of 65 with very marked generalized arteriosclerosis.

These experimental cases demonstrate that a pull exerted evenly through the mesenteric vessels in the presence of increased intra-intestinal pressure produces small traction pouches of the bowel wall very similar to the earliest stages of the multiple diverticula which have been described. It seems probable, therefore, that these two factors are also at work in the production of the multiple diverticula during life.

It seems quite possible that arteriosclerosis of the mesenteric vessels may be a very relevant factor. Not only would this lead to a loss of elasticity of the vessels, thus increasing the traction effect, but the narrowing of the vessels would lead to a decreased blood-supply to the bowel with consequent weakening of the muscle and a tendency to atonia and dilatation. Such dilatation of the bowel is seen at operation in advanced cases, and will be

likely to lead to a slowing up of the passage of bowel contents, both fluid and gaseous, and thereby to a rise in the intra-intestinal pressure.

In the writer's own four cases where tissue was available for microscopy arteriosclerosis of the mesenteric vessels was definitely present in three. In the fourth the vascular tissue available was insufficient for any definite statement to be made on this point as the mesentery had been cut close to the bowel wall.



FIG. 258.—X-ray of first loop of jejunum from experimental case. The lumen of the bowel is filled with plaster-of-Paris cream. The mesenteric vessels are injected with red lead. Tiny traction pouches opposite the attachments of the vasa recta to the bowel wall are clearly visible. In the upper part of the picture the injected vessels showed somewhat faintly and have been emphasized for reproduction.

In so far, therefore, as the writer's own cases supply evidence mesenteric arteriosclerosis seems to be constant, and it would appear that it can act as the initial cause of both factors required to produce the condition—(1) traction by the vessels from without, (2) atonia of the bowel wall with consequent increased pressure from within.

Moreover, mesenteric arteriosclerosis as the initial cause of the condition fits in well with the clinical evidence obtained from an analysis of the histories of the twenty cases on which this paper is based. The average age was 64.

Arteriosclerosis was present to a degree sufficiently marked to attract attention of itself during life in nine of these.

In a total of 11 deaths, the causes were: cerebral degeneration 1, mesenteric thrombosis 1, coronary disease 1, uræmia 2, perforation of a diverticulum 2, pneumonia 3, acute obstruction 1. It will be seen that in the first five of these considerable vascular degeneration must have been present. In at least six of the twenty cases diverticula of the sigmoid were present, and in four chronic constipation was a marked symptom, and it is probable that these facts imply a degree of intra-intestinal pressure above the normal. Where the symptoms are found attributable to the fully developed condition, the most troublesome feature is often a feeling of distension, and there would seem to be no doubt that at this stage the intra-intestinal pressure is certainly raised as an almost constant condition.

Some degree of sclerosis of the mesenteric vessels must, however, be a fairly common occurrence in elderly people, especially those with marked general arteriosclerosis or chronic renal disease, but it is clear that all such cases do not develop diverticula. The explanation of this is probably that even though one factor, the increased traction, may be present, the other necessary factor, that of increased intra-intestinal pressure, fails to develop in many cases.

SUMMARY.

Multiple diverticula of the small intestine at its mesenteric border occur as an acquired condition, being found only in persons who are past middle age.

The writer suggests that the underlying causative factor in this condition is a sclerosis of the vasa recta of the superior mesenteric artery, which thereby exert an abnormal traction effect upon the bowel wall.

Small traction pockets are thus formed consisting of all coats of the bowel, which tend to grow to form large diverticula under any circumstances which increase the intra-intestinal pressure, whether such circumstances are due indirectly to the arteriosclerosis or to other causes.

Having reached a certain size, these diverticula grow much more rapidly, the mucous membrane herniating through the muscular coats.

In the early stages distinguishing clinical symptoms of the condition are usually absent, but in the advanced cases a definite set of symptoms may become manifest.

The condition may at times be diagnosed by X rays. The symptoms may in some cases be alleviated by medical treatment.

Radical surgical treatment is sometimes possible. Acute complications are rare, but occasionally occur, requiring urgent surgical aid.

The greater part of the work for this paper was carried out while the writer was House Surgeon to the Surgical Unit and Senior Surgical Casualty Officer at St. Thomas's Hospital. His sincere thanks are due to all those who allowed him to study cases under their care or placed material at his disposal.

for pathological examination, particularly to Sir Cuthbert Wallace, Mr. C. R. Nitch, Dr. M. A. Cassidy, and Mr. B. W. Williams, of St. Thomas's Hospital; Dr. H. Nockolds and Mr. D. C. Taylor, of Lewisham Hospital; and Dr. W. G. Barnard, late Curator of the Museum of the Medical College, University College Hospital; also to Sir Arthur Keith for helpful advice and permission to study specimens in the Museum of the Royal College of Surgeons. His thanks are also due to Dr. Geoffrey Fildes, of St. Thomas's Hospital, for his great assistance in obtaining the clinical and experimental X-ray photographs.

REFERENCES.

- ¹ BALFOUR, *Ann. of Surg.*, 1913, lvii, 903.
- ² BERRY, *Lancet*, 1927, ii, 496.
- ³ BRAITHWAITE, *Brit. Jour. Surg.*, 1923, xi, 41.
- ⁴ BRAITHWAITE, *Guy's Hosp. Gaz.*, 1918, 77.
- ⁵ CASE, *Jour. Amer. Med. Assoc.*, 1920, lxxv, 1463.
- ⁶ COCKINNIS, *Jour. of Anat.*, 1930, lxiv, 200.
- ⁷ EVANS, *Brit. Jour. Surg.*, 1929, xvii, 34.
- ⁸ GORDINER and SAMPSON, *Jour. Amer. Med. Assoc.*, 1906, xlii, 1587.
- ⁹ GRAZER, quoted by Helvestine.
- ¹⁰ VON HANSEMAN, *Virchow's Arch.*, 1896, cxliv, 400.
- ¹¹ HELVESTINE, *Surg. Gynecol. and Obst.*, 1923, July, xxxvii.
- ¹² KLEBS, *Handbuch f. path. Anat.*, 1869, i, 271.
- ¹³ McWILLIAMS, *Ann. of Surg.*, 1921, lxxiii, 135.
- ¹⁴ TAYLOR and LAKIN, *Lancet*, 1910, i, 495.
- ¹⁵ TERRY and MUGLER, *Arch. of Surg.*, 1921, ii, 347.
- ¹⁶ THOMPSON, *Edin. Med. Jour.*, 1919, xxiii, N.S., 200.

CLEFT PALATE.

BY W. E. M. WARDILL.

ASSISTANT SURGEON, ROYAL VICTORIA INFIRMARY, NEWCASTLE-UPON-TYNE.

(Being a Hunterian Lecture delivered before the Royal College of Surgeons of England on January 27, 1933.)

THE results of the treatment of cleft palate have left so much to be desired that a great many, disheartened at their efforts, have slumped the subject rather than admit their after-results. It is almost axiomatic that the lack of success in the treatment of any complaint is in direct proportion to the number of remedies, and the truth of this is amply illustrated in cleft palate by the number of methods which appears from time to time for its treatment. But from the morass of literature which has sprung up, one fact seems to be generally recognized—namely, that any treatment must aim at the restoration of contact between the posterior surface of the soft palate and the pharyngeal wall, whereby complete separation of the nasal and oral cavities can be effected. The ingenious method of Gillies and Fry was amongst the first devised with this aim.

THE MECHANISM OF SPEECH.

During normal speech, the palate mechanism is kept in a state of constant activity, the palatopharyngeal valve opening and closing at an extraordinarily high speed. Most of the consonants require closure of this valve, the exceptions being the *m*, *n*, and *ng*, where sound, and the air current, are allowed to escape through the nose. The remainder of the consonants are all explosives in that they require complete separation of the two cavities, so that air, voiced or unvoiced, should be pent up in the oropharyngeal cavity during or prior to the pronunciation. The *b*, for instance, requires 'voiced air' to be compressed before the final explosive effort which separates the lips and thus results in the production of the sound; *p* is made in the same way, with the exception that the pent-up air is not 'voiced' by the larynx. On the other hand, *m*, *n*, and *ng* require an open palatopharyngeal valve in order that the nasal resonance should be added. It will thus be admitted that destruction of the palatopharyngeal valve will place the possessor in an extraordinarily disadvantageous position in that the only sounds it is possible to produce correctly are the nasal resonants, as, of course, every attempt at the imprisonment of air for the explosive effort of the consonants is foiled by escape through the nasal cavities. The reverse of this is well seen in the common cold, where nasal obstruction converts the *m* into a *b*, the *n* into a *d*, and the *ng* into the hard *g* as in the well-known advertisement for 'Colbad's bustard' (Colman's mustard).

Actual closure of the palatopharyngeal valve is executed by two sets of muscles—those which move the palate and those which act on the pharynx. The muscles moving the palate are of two kinds—those which draw the palate to the pharyngeal wall and those which oppose this action and cause the palate to be drawn from the pharyngeal wall. Of the former, the levatores of the palate are the only ones of importance; they cause the soft palate to arise in a comma-like shape and make contact between its upper surface and the pharyngeal wall at about the level of the anterior arch of the atlas. Damage to, or destruction of, these levatores is the greatest disaster which can happen during any operation for the repair of the palate. The muscles causing withdrawal of the palate from the pharyngeal wall are those which enter into the pillars of the fauces—namely, the palatoglossus and the palatopharyngeus, and to a very small extent the tensor palati.

The other or pharyngeal half of the palatopharyngeal valve is formed by the upper part of the superior constrictor muscle of the pharynx, which, to effect closure of the valve, contracts strongly and produces a well-marked ridge or cushion on the posterior pharyngeal wall, at the same time narrowing the nasopharynx from side to side. This muscular action can be readily observed in any case of unrepaired cleft of the palate during the pronunciation of 'Ah' with the mouth wide open. It was first described by Passavant, and his name is usually attached to its description. In addition to causing this narrowing of the nasopharynx, the cushion of Passavant also causes the two halves of a cleft soft palate to be approximated, and recognition of this fact led me to prophesy the existence of fibres of the muscle passing into the soft palate and not terminating, as in the standard descriptions of the anatomical text-books, in bony or ligamentous structures. Dissections proved this prophecy to be correct and the observation was amply confirmed by my colleague, Dr. James Whillis, and has been more fully described by him in the *Journal of Anatomy*.¹ The importance of the observation lies in its proof that the superior constrictor is in fact a palatal muscle and therefore part of the palatopharyngeal sphincter mechanism.

DEFECTS IN THE NASOPHARYNX IN CLEFT PALATE.

In a previous Hunterian Lecture,² measurements taken by myself from a series of normal and cleft-palate skulls were quoted to show that in cleft palate there is a marked increase in the bony diameters of the nasopharynx, and that any operation designed to restore the palatopharyngeal valve must take account of this, otherwise the palate might be repaired with the best possible æsthetic result yet no closure of the palatopharyngeal valve attained. It was shown that the hamular processes which act as pulleys for the tensors of the palate were widely separated, and that if they remained intact the pull of the shortened muscle was sufficient to prevent suture without tension and with consequent immobility of the soft palate. Furthermore it was recognized that, the muscle being shortened, the effect of putting tension on the palate from side to side was to cause further shortening in an antero-posterior direction. To overcome this difficulty it was advised to destroy

the hamular processes.* But as all the diameters of the nasopharynx are increased, mere reduction in the tension from side to side is not likely to overcome this deficiency, and for this purpose the operation of pharyngoplasty was devised.

PHARYNGOPLASTY AND REPAIR OF THE PALATE.

This operation has for its object the imitation and exaggeration of the normal method of palatopharyngeal closure—that is, an artificial, but greatly exaggerated, cushion of Passavant is created on the posterior pharyngeal wall. The method of carrying it out is similar in principle to the Miekulicz pyloroplasty, with the difference, of course, that the operation is designed to narrow and not to widen the channel. Briefly, a transverse incision is made on the posterior pharyngeal wall and passes through the superior constrictor muscle. The incision is then sutured in a vertical direction, thus effecting reduction in the volume of the nasopharynx.

Anatomical Considerations.—The superior constrictor muscle lies immediately beneath the pharyngeal mucosa. Its posterior surface is closely enveloped in a thin but tough aponeurosis, the buccopharyngeal fascia, which separates the muscle from the loose connective tissue of the retropharyngeal space. It is extremely important to recognize this relationship, since the buccopharyngeal fascia protects the retropharyngeal space from perforation during the course of the operation. At each side of the nasopharynx a strong fold of fibromuscular tissue passes vertically downwards from the Eustachian tube and the base of the skull and encloses in its substance the salpingopharyngeus muscle. Recognition of this bundle is very important, since it must be included in the sutures used to narrow the nasopharynx. Deep to these salpingopharyngeal folds there is a fair amount of loose connective tissue, the buccopharyngeal fascia not investing the fibres of the superior constrictor so closely as on the posterior pharyngeal wall.

Technique of Pharyngoplasty.—

Anæsthesia.—The best anæsthesia for all palate work is endotracheal gas-oxygen and ether administered by the method invented by I. W. Magill. I would like to express my personal gratitude to Dr. Magill, who first demonstrated the method to me, and to Dr. W. J. Phillips, of Newcastle, for having taken the trouble to master the technique.

One great advantage of this method of anæsthesia is that bleeding and excretion of mucus do not interfere with the airway, and the operation can be carried out in a deliberate fashion without the constant aspiration which seems to occur with any of the other methods. Moreover, a long operation is suffered by even a young infant without any material deterioration in the general condition. At first the endotracheal tube was a nuisance, but with practice it can be tucked away to the right of the mouth at the side of the tongue.

* Destruction of the hamular processes was, I believe, first advocated by Billroth. I was in complete ignorance of this when I arrived at the same conclusion as the result of my anatomical studies.

The Operation.—Using a headlight, the operation is carried out in a semi-darkened theatre. The patient is anesthetized and the head thrown well back. A gag is placed in the mouth, the tongue being well depressed. By far the most useful gag is the Boyle-Davis as modified by Mr. Norman Dott, of Edinburgh. Mucous is swabbed away, using small gauze mops wrung out of 1–150 carbolic lotion.

The halves of the soft palate are held aside by small sharp Gillies' hooks and the nasopharynx is then lightly stimulated to produce reflex contraction of the pharyngeal muscle and to mark the level of the ridge of Passavant. Using a sharp-pointed knife, a transverse incision is made through the mucosa and the superficial fibres of the superior constrictor muscle at about the level of the ridge of Passavant. By means of a special dissector, really a standard aneurysm needle cut short, the constrictor muscle is gently peeled from the surface of the buccopharyngeal fascia in both an upward and downward direction until a considerable area is undermined. The peeling process is continued laterally right up to and beyond the salpingopharyngeal folds, and, using the dissector as a retractor, the incision is continued laterally up to these folds. During the process of dissection, care must be taken that the buccopharyngeal fascia is not perforated, since, although no trouble has arisen from this cause, it is not considered advisable to open up the retropharyngeal space with all its risk of infection. One can be certain of the fact that this space has not been perforated so long as resistance to the peeling process is encountered.

The business of suturing is now commenced, and using No. 0 catgut on a small Reverdin needle a suture is carried through the salpingopharyngeal fold on each side. It is extremely important to obtain a big bite of tissue and a firm hold, and on occasion the extreme edge of the upper surface of the soft palate has been included in this suture. The suture is not tied but is held with an artery forceps and acts as a very excellent retractor. Further sutures are then placed above and below the first until in all from three to five have been inserted. The remaining sutures, while used as a routine, are not felt to be of any great moment, but merely secure the coaptation of the edges of the mucous membrane. The sutures are now tied and cut short. It is impossible to make a really neat and tidy job of pharyngoplasty, but perhaps this is fortunate in that there is less likelihood of locking sepsis in a notoriously bad area. When the operation is completed it will be observed that the two halves of the soft palate lie much nearer the middle line and very often the halves of the uvula overlap.

There is no need to do any preliminary operation either for the removal of tonsils or adenoids, although the latter, if unduly prominent, may be a nuisance in that they interfere with the uppermost suture of the pharyngoplasty, or may give rise to some troublesome oozing of blood, thereby helping to obscure the field. The most important point to observe in this operation is the securing of a very firm hold of the salpingopharyngeal folds, since within a few days the sutures tend to soften and may come out before they have fulfilled their object. Difficulties are often encountered, particularly in infants, and mostly because of the small size of the parts and the extremely restricted area in which the operation is carried out. Once the

pharyngoplasty is completed, it behoves the anæsthetist to keep a sharp look-out for any sign of respiratory obstruction.

At this stage any further procedure can, if thought wise, be abandoned, but it is now my invariable practice to go straight ahead and repair the palate, since suture of the soft palate on top of a pharyngoplasty lends additional support to the latter, and at the point where the need is greatest—namely, at the insertion of the outermost sutures through the salpingopharyngeal folds. Moreover, the pharyngoplasty helps to reduce the tension on the sutures in the soft palate.

One criticism launched against this operation is the statement that a successful pharyngoplasty is not likely to remain in this condition, and that the artificial narrowing will disappear and the parts return to the normal state of affairs. Observation of many cases over a period of the last seven years has shown that this does not occur. Apart from what happens within a few days of operation, no widening or giving of the nasopharyngeal part of the operation has ever been observed.

Complications and Dangers of Pharyngoplasty.—In my original paper published in 1928 mention was made of certain dangers of the operation. The chief amongst these was the risk of retropharyngeal cellulitis, and reference was made to a case in which this might possibly have been a cause of death, although the post-mortem evidence still left one in considerable doubt. In all, the operation has been performed on seventy-two patients without any sign of recurrence of this trouble, and this in spite of the fact that it is believed that, owing to careless dissection, the retropharyngeal space has been opened on two or three occasions. Even so, this statement should not lead to the belief that such an accident will always go unpunished.

One risk which is ever present after this operation when combined with repair of the palate is that of asphyxiation during the course of recovery from the anæsthetic, and the more successfully the operation has been done the greater the risk. The aim of the procedure is to restore a palatopharyngeal valve, and in order to be successful it is necessary to over-correct, and a great many of these cases at the end of the operation are quite unable to breathe through the nose, necessitating continued traction on the tongue until recovery is complete. Latterly in using Magill's method of endotracheal anæsthesia, it has been our custom to leave the tube in the trachea until reflexes are fully restored.

Deafness was a further complication mentioned in my previous communication, but this has not turned out to be the boggy expected. Several cases have had temporary deafness which has cleared up in a few days; in no case has it remained permanent. My experience has been that sufferers from cleft palate, whether subjected to operation or not, are more liable to this trouble than the normal person—presumably owing to irregularities in the Eustachian tube or from its increased exposure to infection.

Repair of the Palate.—In repair of the palate, the method used by myself embraces what I believe to be the best in the Langenbeck-Fergusson and the Veau operations.

Using the left index finger the position of the hamular process is located, and beginning outside this point a lateral incision is made and carried forward

on to the hard palate close to the alveolar margin. This procedure is carried out even where the cleft is of the soft palate only. By means of blunt dissection the hamular process is defined, and is then completely detached from its base either by cutting it with strong scissors or wrenching it with an elevator. Careful palpation will make certain of the complete separation of the process. This procedure is then carried out on the opposite side, and it will be observed that immediately after successful destruction of the hamular processes the two halves of the soft palate fall much more closely together in the middle line, and also fall backwards towards the pharyngeal wall. No regrets need be felt at the apparently wanton destruction of bone, since it has as its main object the complete freeing of the shortened tendons of the tensors of the palate. The tensors undoubtedly occupy the key position in that they prevent movement of the palate and must be destroyed at any cost.

Inserting an elevator into the lateral incision, the mucoperiosteum of the hard palate is gradually stripped up from the bone; the stripping being carried forward as far as the anterior end of the cleft and the lateral incisions enlarged to the same extent. The separation is then carried out along the edge of the cleft and at the junction of the nasal and oral mucosæ. Then, passing an elevator either through the lateral incision or beneath the mucoperiosteum in the borders of the cleft, the soft palate is separated from the posterior edges of the hard palate. This is carried out entirely without further incision of the mucous membrane, and it is not necessary—indeed, it is regarded as highly inadvisable—to make any incision through the nasal mucosa at this point. The use of right-angled scissors as in the Langenbeck-Fergusson operation is mentioned only to be condemned, as it is believed that this act, which cannot be carried out with precision, is likely to cause damage to the levator muscles. Moreover, submucous separation means that less raw area is left to granulate, and the chances of infection are consequently materially reduced.

It should now be found that the two halves of the separated palate will meet in the middle line and will lie comfortably against the raw bone from which they have been separated. Should this not be so, the lateral incisions are extended further forward and then carried inwards to the cleft so that the whole of the mucoperiosteum of the hard palate is lifted up as a flap, as in the method of Veau. It will then be found that this flap can be slid inwards and sutured in the ideal position, lying snugly against the bone and without tension. One great advantage of raising flaps is that they can be turned backwards so that the greater part of the separation of the soft palate from the hard can be further assured under the guidance of the eye. Furthermore, the posterior palatine artery with its fascial covering can be loosened from its bony canal, thereby allowing a little extra inward movement of the palate. A great deal has been written about the necessity of preserving this vessel, and while I would not advocate its division as a routine procedure, on the occasions when I have done this by accident or design I have seen no ill results following. Sometimes it is essential to divide both vessels so as to bring the two halves of the palate together without tension, and often further freeing can be effected by dissection in those parts of the lateral incisions lying behind the fractured hamular processes.

To complete the repair of the palate, the nasal mucosa is separated from the upper surface of the bone and in the suitable case a vomerine flap can be turned aside as in the method advocated by Veau. My own experience has been that while I subscribe absolutely to the soundness of Veau's opinions on this procedure, I have found that the very cases in which I most wanted to carry out his methods have been the very ones in which it has not been completely possible. At any rate, wherever practicable, the nasal mucous membrane covering the upper surface of the hard palate is sutured in the middle line. This principle of suturing the nasal mucous membrane with fine catgut is carried right along the whole length of the hard and soft palates as far as the tip of the uvula, having first of all, of course, pared the edges of the soft palate. The operation is completed by using fine silkworm sutures for the under surface of the soft palate and the mucoperiosteum of the hard palate. The Veau 'suture musculaire' has not been used in my cases.

Mortality.—There seems to have been some hesitance on the part of general surgeons to adopt this operation, and on grounds believed to be associated with an expected mortality. In all I have done pharyngoplasty on 72 patients, with a total mortality of 3. The first case occurred amongst the earliest of the series, and death took place within thirty-six hours of operation; the other two cases were the direct result of the anæsthetic. It is worth while examining these cases in detail.

Case 8.—A boy, aged 4 years, who was submitted to pharyngoplasty without repair of the palate. During the course of the operation it was noted that the stripping of the superior constrictor muscle was carried out without resistance, and it was not recognized till too late that the retropharyngeal connective-tissue space had been opened. The operation was completed uneventfully, but the child died thirty-six hours later from what was believed to be acetonaemia, although at the autopsy a slight degree of retropharyngeal cellulitis was present and a very thin purulent track extended from the site of operation to the mediastinum. This case was only the eighth upon which the operation had been done and it caused very close examination of the operative procedure. Since that date the retropharyngeal space has been accidentally perforated on several occasions, but there has been no recurrence of the trouble.

Case 78.—A girl, aged 6 years, who had had diphtheria about eight months before operation. A preliminary tonsilleotomy was done, and after waiting about six weeks, repair of the palate was undertaken. The anæsthetic was chloroform and ether mixture administered by a student anæsthetist. Pharyngoplasty was performed, and during subsequent repair of the palate it was observed that bleeding suddenly ceased, and the child died. This case was a great lesson in that it proved that anaesthesia in these cases is very difficult and can only be carried out in reasonable safety by anæsthetists of great skill and experience. The student in this particular case had given many anæsthetics and was in my opinion a born anæsthetist, but it was undoubtedly unfair on my part to ask him to undertake a case of this nature.

Case 53.—Aged 3 years. Endotracheal gas-oxygen and ether was given by the method of Magill, and pharyngoplasty and repair of the palate were done in one stage. The operation presented no unusual difficulties, and at its completion was regarded as entirely satisfactory. The pharynx was mopped dry and the endotracheal tube removed and the child returned to bed. A few minutes later an urgent message from the ward caused an immediate visit to be paid, and it was seen that the child was obviously asphyxiated. The airway seemed to be completely free, but it was certain that the child had drowned in its own secretions. Whether

these secretions were from the stomach and resulted from vomiting, or whether they had been retained behind the soft palate and only appeared on removing the patient to bed, was never satisfactorily explained, but the lesson learnt was one which has since proved extraordinarily useful; the anæsthetic tube is now never removed until the reflexes have returned and until it is certain that no secretions remain dammed up behind the soft palate.

Pharyngoplasty, with or without repair of the palate, has, then, in my hands given an avoidable mortality of 4.1 per cent, and the question arises whether the results justify this loss of life. Unhesitatingly I say 'Yes', and especially in view of the fact that the operation is new and, as yet, experimental. Cleft palate is a deplorable deformity for those who are unfortunate enough to possess it, and very much greater risks than those outlined above would not prevent me from advocating my present methods. The reaction of one's medical friends in this respect is most illuminating; when they are faced with the problem of cleft palate in their own offspring they say: "We appreciate the risks, but operate, operate early, take plenty of time, and make a sound surgical job."

Consideration of the three deaths quoted above shows that in no case was death due to inherent defects in the operation, but only to the occurrence of avoidable accidents. But apart altogether from the mortality of any operation, it will be agreed that it would be hardly justifiable to take the risk of carrying it out if the patient were to hang between life and death and only recover after a prolonged convalescence. This has not been my experience. It nearly always happens that after operation there is some reaction in the form of a rise in temperature, and on rare occasions the temporary enlargement of cervical glands has been noted. One sickly child, aged 13 months, gave reason for some anxiety for about a week; it was fevered, and the palate never showed any healthy reaction; most of the sutures ultimately gave way, but the child recovered. Delay in attempting operation in this case would have been advisable.

Latterly, the choice of the right time to operate on infants has been greatly facilitated by the skilful medical supervision of Dr. J. C. Spence and other members of the staff of the Newcastle-upon-Tyne Babies Hospital.

THE RESULTS OF OPERATION.

The results of a new operation merit very careful study before a strict valuation can be placed upon them. First of all it is necessary to recognize that from a surgical standpoint all that can be obtained is the formation of a functional valve separating the oral and nasal cavities. No operation has yet been devised which will make the possessor of such a valve either speak properly or use it for the other functions for which it is required. In any discussion of the results, therefore, it necessarily follows that there are two criteria of success: first, the restoration of the palatopharyngeal valve; and, secondly, the restoration of normal speech. If the first criterion is attained, it does not necessarily happen that the second will also be attained, but it is quite certain that if the first criterion, the formation of a competent palatopharyngeal valve, is not attained, then the second, of normal speech, cannot possibly follow.

Restoration of the Palatopharyngeal Valve.—The question arises of how one is able to prove the existence of a competent palatopharyngeal valve. Peroral inspection of the valve is almost useless, and is a very unreliable guide as to what is taking place behind, since contact between the palate and the pharyngeal wall occurs at the level of the anterior arch of the atlas and at a considerably higher level than the uvula. This was forcibly brought home to me in one of my cases where it would have appeared from inspection that closure of the pharyngeal valve might be impossible, yet the patient was able to present proof positive on trying the various tests of function. Equally, other cases have been seen in which it would appear certain that the valve was competent, yet on further trial it was proved without doubt that palatopharyngeal closure was not being effected.

Fortunately there are a number of simple tests which are of very considerable importance and if applied properly are believed to be accurate; they can easily be carried out in any consulting-room. Of these the 'snorting test' is by far the simplest. The patient is instructed to put the tongue slightly out and grip it between the lips. Air is then sucked into the nose as in the impolite act of clearing away pharyngeal mucus. A characteristic sound closely resembling the snore of a deep sleeper is produced and can be sustained and repeated if pharyngeal closure is being attained. The reason for protruding the tongue and closing the lips is to prevent accessory noises being formed behind the tongue. Various degrees of snorting are recognized, and one fallacy is seen in some cases where closure can be maintained for just a fraction of a second, the valve only being competent with severe muscular effort. It is interesting to observe that snorting is sometimes obtained in cases where the soft palate is almost completely immobile, and presumably is due to the contraction of the pharyngeal muscle and possibly also to the slight pull of the levatores. Snorting is a natural attainment of the normal individual and is seen at its best in the presence of a chronic pharyngitis. It is an impossible feat in the case of the unrepaired cleft palate, a fact often reflected in the dirty state of the pharyngeal mucosa in such cases. It is equally impossible in the large majority of cases submitted to operation by the classical methods, and casts a great reflection on their results. As a means of assessing the efficacy of operation on the palate it is of inestimable importance. Holes in the hard palate do not alter its value, since the tongue effectively seals them and prevents air entry by any other way than through the nasopharynx.

A rough and somewhat inefficient test which can be carried out even in the presence of an incompletely repaired hard palate is the ability of the patient to blow out a match or a candle. Improvement in performance can be measured by the increased distance at which this is possible. A great fallacy is, of course, the degree of nasal obstruction which might exist and give a false impression of palatal closure.

Rubber balloons are very popular with children, and usually require such a degree of expiratory effort to inflate them that the feat becomes impossible if holes exist in the palate or if the nasopharyngeal valve is incompetent. As a test of strength it is excellent. One child, however, succeeded in evading the test by pursing the lips to such an extent that they overlay the nostrils

and so prevented nasal escape. A second child succeeded in inflating a balloon by using a mouthful of air at a time, the air being imprisoned between the tongue and the roof of the mouth and forced into the balloon by the pressure of the tongue, the lips being tightly closed on the rubber outlet between each effort. In the absence of these tricks, however, the test is reliable.

A further test is by the use of the nasal irrigator or aqueous spray, and is only applicable to adults or older children. The head is thrown back and a bland fluid (saline or weak antiseptic lotion) is introduced into one nostril until it begins to return through the other, a feat easily accomplished in the normal person. If the nasopharyngeal valve is incompetent, a reflex disturbance of a major sort is induced and the experiment terminates abruptly; consequently this test can only be classified as confirmatory.

Another possible test consists of connecting an airway between one nostril of the patient and the ear of the examiner. The apparatus consists of a rubber tube having at one end a glass nozzle to fit the nostril of the patient and at the other the ear-piece of a stethoscope. The patient is then instructed to say any of the consonants, with the exception, of course, of the nasal resonants (*m*, *n*, or *ng*). Nasal escape is accurately detected by the ear of the examiner. For this idea I am indebted to Sir Richard Paget.³

A more complicated method was demonstrated by me at the Royal Society of Medicine in June, 1927. Separate tubes are introduced into the mouth and the nostrils, the latter being Y-shaped and completely filling both nostrils. The nasal and oral escapes are collected in separate gas-bags and can be measured. In use, the patient is instructed to blow through the mouth tube without allowing nasal escape, an impossible feat if the palatopharyngeal valve is incompetent. The amount of air collected in each bag is then measured and the degree of incompetence can be estimated. By a simple mathematical calculation the cross-section area of pharyngeal deficiency can be arrived at, although it is admitted that there is a very large degree of experimental error.

So much for the surgical problem of the formation of a functional valve and the proofs of success. Before passing to the second criterion of restoration of normal speech further important factors require consideration, and particularly those responsible for the production of cleft-palate speech.

Causes of Cleft-palate Speech.—A child born with a cleft palate wakens to a sense of its deficiencies in the first few hours of life when it realizes that the business of feeding is not so easy as it might be. In the complete cleft, sucking the breast or the bottle is an impossible feat unless the nostrils are at the same time occluded, and deglutition and aspiration must at first be extremely difficult to distinguish. These problems surmounted, the business of speech is the next in order, and the child's first effort to communicate with its fellows—namely by crying—offers no serious difficulty. The first word uttered by most infants is 'Mamma', and as the consonants of the word are nasal resonants and require an open palatopharyngeal valve this is easily accomplished. But when the second word 'Dadda' or 'Gagga' is attempted, the incompetence of the nasopharynx becomes a real problem. If the hearing

is acute and the speech sense good, the cleft-palate infant will recognize that there is only one way of saying 'Dadda'—namely, by placing the tip of the tongue against the roof of the mouth immediately behind the alveolar margin and then allowing the explosive effort to take place as the tongue is suddenly withdrawn. If this realization of the physical disabilities of speech is clearly brought home, the child will grow up using the tongue and palate, making the consonants in the correct way and in spite of nasal escape. The task will be rendered the easier the less the degree of the cleft, but it does not necessarily follow that the less the degree of the cleft, the better will be the speech.

Such cases as those outlined above are not uncommon in cleft-palate practice and offer by far the best type on which to attempt operation, and for purposes of classification have been labelled *Group 1*.

Let us imagine a second group of cleft-palate speakers. In *Group 2* the child has a fair degree of acuity of hearing and is a trier. It attempts the big stumbling-block, 'Dadda', and finds that nasal escape prevents reasonable expression. It looks round for a means of overcoming this nasal escape and finds that movement of the muscles controlling the *alæ nasi* assist in preventing this. Herewith is born one of the most characteristic of the articulatory gymnastics of the cleft-palate speaker, and one which very often remains long after the palate has been successfully repaired, but fortunately does not interfere materially with speech training. It takes the form of violent occlusive movements of the *alæ* of the nostrils during speech, but although it may assist in reducing nasal escape it does not succeed in removing the stigma of cleft palate. This observation can be easily verified by nipping the nostrils of any cleft-palate speaker, when it will be observed that the type of speech does not alter although it may be somewhat modified. Such a child will often have difficulty with the *s*, and discovers that this sound, instead of being made between the tongue and the teeth, can be formed in quite a reasonable way in the pharynx, and thus is born another of the characteristic cleft-palate sounds which give so much trouble in re-education. The sound is not always easy to recognize, as it can be made in such a skilful fashion that it resembles fairly closely the normal *s* sound. Its recognition in cases of doubt is, however, made simple because the cleft-palate *s* can be made with the mouth wide open, a feat which is impossible in the normal *s*. The consonants, with the exception of the nasal resonants (*m*, *n*, *ng*), require a closed nasopharyngeal valve and in all the oral air-stream is partially or completely interrupted in some part of their formation, and it is at this point that the glottic stop is introduced as a means of disguising the absence of adequate oral interruption. It is used by completely closing the glottis and placing tongue, teeth, lips, etc., in their more or less correct relations and then suddenly releasing the glottis to allow of the escape of air and the formation of the sound. In this way a very fair imitation of many of the consonants is possible, but such speakers are mostly difficult to understand. (The glottic stop is occasionally used in the English language where two vowel sounds meet as in 'the ether'.)

In *Group 3*, or worst group, where one presumes that acuity of hearing and intelligence are at their lowest, it may be that not even the trick of the

glottic stop is acquired and all the sounds resemble those of vowels. Such speech, except to the ears of love as represented in the patient's nearest and dearest, are completely unintelligible. But it is not fair perhaps to think that all such are untrainable.

Consideration of the above factors in the causation of cleft-palate speech provides the clue to the reason for the failure after any operation however carefully performed. If the child is of the type mentioned in the first group, where the speech mechanism is brought into correct anatomical position for the formation of each sound, then the supply of an adequate or even slightly incompetent palatopharyngeal valve can be expected to result in perfect speech, or in speech that is nearly perfect. If the cleft-palate gymnastics have been thoroughly acquired, no operation of any sort can possibly help without speech training in which these tricks are unlearned and replaced by normal methods. It is useless, therefore, to classify cases according to the speech results alone, since these merely indicate the proportion of ambitious patients and the skill of the speech trainer. Wherefore in the consideration of the results of operation my cases have been divided into four main groups:—

1. Firstly, those who possess the prime factor of importance—namely, a competent palatopharyngeal valve—and who therefore have the functional physiological mechanism for the production of normal speech;

2. Secondly, those who speak without any cleft-palate stigma and are therefore, apart from their dialect and other peculiarities, perfect speakers;

3. Thirdly, those who have neither normal speech nor a physiologically competent valve; and

4. Fourthly, those who are too young to apply the various tests or to make certain of their condition by their speech; also cases in which the operation is of recent date.

As mentioned previously, the operation of pharyngoplasty has been done on 72 patients; 3 of these died; 3 have not been traced; in 10 the operation was done as a secondary procedure and will be considered later; and in one case, although the pharyngoplasty was done about two years ago, repair of the palate has not yet been completed. This leaves 55 patients for consideration.

Discussion of Results shown in Table I.—The cases recorded above have not been chosen in any way either as to age or type. Many had had previous operations by other surgeons. They represent all-comers, and I do not recall ever having refused to operate excepting in one case with diabetes. As will be observed, the age of operation varies between 6 months and 45 years, and can be accepted as the usual run of an average surgical practice. It will be noticed, however, that only three of the cases classified as possessing competent valves have been submitted to operation before the age of fluent speech, whereas amongst the group of perfect speakers are only three who had passed this age.

Some of these cases are worth considering in further detail in relation to their respective groups.

Taking first those where valves have been successfully formed: *Cases* 16, 24, 28, 62, 79, and 5 all possess excellent palatopharyngeal valves, and

although they all belonged to the *Group 2* of cleft-palate speakers, as a result of training, either professionally or by their parents, they are able to obtain every sound in normal speech and to pronounce each sound without any fault whatever, yet they cannot be transferred to the group of perfect speakers, since, if they are taken unawares, the speech immediately develops the characteristic stigmata. In none of my cases is this allowed to pass as perfect speech, although I feel that I should be justified, since obviously the valve mechanism has been successfully restored and moreover the patient can speak perfectly at will.

Table I.—RESULTS IN FIFTY-FIVE CASES OF CLEFT PALATE.

COMPETENT VALVES			NORMAL SPEECH			DEFECTIVE SPEECH AND NO VALVE			TOO YOUNG TO ASCERTAIN OR OPERATION TOO RECENT		
Initials	Case No.	Age at Op.	Initials	Case No.	Age at Op.	Initials	Case No.	Age at Op.	Initials	Case No.	Age at Op.
		Years			Months			Years			Months
L. G.	75	3	B. S.	34	6	G. W.	15	2½	J. R.	47	11
L. G.	60	3½	E. S.	31	7	J. A.	76	11	J. W.	67	11
W. G.	71	3½	J. B.	29	11	W. P.	1	17	A. G.	58	12
H. B.	69	6	H. H.	57	25	A. E.	41	19	D. H.	21	13
A. S.	62	7	E. B.	14	26	S. C.	2	24	J. W.	27	14
T. W.	20	7	J. D.	59	27	D. S.	9	45	C. P.	45	14
J. B.	11	8	J. L.	52	31				A. R.	93	21
J. W.	19	8	V. D.	48	34						Years
N. M.	7	9			Years				M. D.	98	2
T. B.	12	10	J. T.	13	3				J. P.	66	2
R. S.	79	13	M. T.	40	3½				J. B.	80	2
G. M.	3	13	P. Mc.	22	7				H. M.	37	2½
E. F.	6	15	J. J.	18	23				J. G.	68	2½
G. W.	82	17	E. S.	80	28				J. A.	91	2½
W. K.	16	18							E. S.	83	3
Mrs. S.	5	26							O. N.	84	3
E. F.	24	32							N. S.	61	7
G. B.	28	34							A. McQ	99	18
D. S.	4	36									

Case 75 was taken to the Antipodes before the end-result of this operation was seen by myself; he was able to snort in my presence, proving his possession of a valve, and shortly after his operation his mother stated that he could say many words with perfect clearness, although this latter was not sufficiently demonstrated to me. It is possible that he may by now, after the lapse of four years, be able to speak perfectly.

Case 20 presented a most unusual state of affairs. He had a cleft of the hard and soft palates, and was submitted to pharyngoplasty followed by repair of the palate. The palate broke down, and two further attempts resulted in failure in that the soft palate gave way along almost its entire length with the exception of about half an inch at the posterior extremity. Abject despair of ever succeeding in this case was only natural, but the boy turned up some twelve months later with the enormous holes completely closed and a freely mobile palate. Moreover he can blow up a balloon—for a child the hardest of all functional tests: speech has improved immeasurably;

the remaining defects are slight, and he is young enough to have these remedied by a short course of training.

In the second class—namely, the perfect speakers—are many cases that are worthy of note :—

Cases 13, 14, 29, 31, 34, 40, 48, 52, 57, and 59, were all submitted to operation either before speech had begun or shortly afterwards. In every case the only speech training has been given by the mother, and of course one finds that mothers vary immensely in their capabilities in this direction. It has been very profitable to me to see these cases at fairly frequent intervals and observe the development of the speech habits. Many of them have had baby talk which has been obviously cleft-palate in type, but almost invariably they have, early in their careers, shown evidence of the presence of a palato-pharyngeal valve in a curious sort of snore executed at intervals whilst attempting to form words. Gradually the snore has disappeared and the stigmata of cleft-palate speech have disappeared with it, until the only remaining fault has been with the double consonants, which appear to give exceptional difficulty. In the course of time even this has been remedied, until now no defect in speech is observable. Some of these cases have not yet got over their baby talk, but their loquacity has enabled one to be certain that there is no stigma of cleft palate, nor is such likely to develop.

Case 18, a woman aged 23 years, had a cleft of the soft palate which reached, but did not involve, the hard palate; she was a *Group 1* speaker and formed all her consonants in the correct fashion but was extremely difficult to understand because of the gross nasal escape. Pharyngoplasty and repair of the palate were done in one stage. After removing the sutures about the tenth day, the patient was made to read from a book, and with a little correction at first she was able to make all her sounds perfectly. She could snort strongly from the very first. This young woman is now, three years after operation, a perfect speaker and does not present the slightest stigma of cleft palate. She has had no training beyond the demonstration given after removal of the sutures.

Case 80, a woman aged 28 years, had a cleft of the soft palate almost reaching the hard. Before operation she spoke with gross nasal escape but did not possess any of the usual articulatory faults, and therefore belonged to *Group 1*. Her operation was performed to enable her to earn a livelihood in Australia and it was expected that she would leave within a week or two of being discharged from hospital. Little interest was therefore shown in her after-progress, but to my surprise she came to see me four years later, and not knowing her again, owing presumably to changes in female fashions of dress, it was only after protracted conversation that I was able to ascertain that she had come to show off her palate. She had not had training, but her speech was just that of any ordinary person. She was unable to inflate a balloon, but could snort in a most convincing manner.

Case 22 is the most remarkable result in the whole series and is recorded in the section dealing with prognosis.

Amongst the bad results are recorded cases which are still under treatment but in which no evidence has yet been adduced to show either a palato-pharyngeal valve or perfect speech, although I am sanguine of effecting at

least the formation of the valve in all. *Case 41* is a mentally dull girl aged 19 years, who had a very wide cleft of both hard and soft palates with little tissue to spare. Operation was most successful in that the cleft was completely closed and the soft palate freely mobile, but no evidence of the restoration of the valve has yet been produced. *Cases 1, 2, and 9* were amongst the earliest of my series and are recorded in a previous Hunterian Lecture.²

'Too young to ascertain' results include a number of cases where operation has been performed in the last few months or where the child has not yet spoken sufficiently to warrant the speech being classified either as normal or definitely cleft-palate in type. *Cases 27, 45, and 60* speak but little, some of the sounds being normal in character and others of the cleft-palate type; but in view of previous experience with regard to the development of speech in other children of this age, it is not felt possible to assign them to any other group, although there is some evidence that they are capable of palato-pharyngeal closure.

In *Table II* the cases are classified according to the type of cleft, and it will be noted that most of my cases have involved both soft and hard palates or have been complete unilateral clefts with hare-lip.

Table II.—TYPE OF CLEFT IN FIFTY-FIVE CLEFT-PALATE CASES.

RESULT	SOFT PALATE		SOFT AND HARD PALATE		UNILATERAL COMPLETE		BILATERAL	
	Initials	Case No.	Initials	Case No.	Initials	Case No.	Initials	Case No.
Competent valves	G. M.	3	N. M.	7	L. G.	75	J. B.	11
			T. W.	20	H. B.	69	W. G.	71
			R. S.	79	A. S.	62		
			E. F.	6	T. B.	12		
			E. F.	24	W. K.	16		
			J. W.	19	Mrs. S.	5		
					G. B.	28		
					D. S.	4		
					L. G.	60		
					G. W.	82		
Normal speech	J. T.	13	B. S.	34	H. H.	57		
	M. T.	40	E. S.	31	E. B.	14		
	J. J.	18	V. D.	48	J. D.	59		
	E. S.	80	P. Mc.	22	J. L.	52		
			J. B.	29				
Defective speech and no valve			W. P.	1			G. W.	15
			W. E.	41			J. A.	76
			S. C.	2				
			D. S.	9				
Too young to ascertain result or operation too recent	J. W.	67	J. W.	27	D. H.	21		
	J. B.	89	J. R.	47	A. G.	58		
	A. R.	93	C. P.	45	N. S.	61		
			H. M.	37	J. G.	68		
			J. P.	66	O. N.	84		
			E. S.	83	J. A.	91		
			M. D.	93				
			A. McQ.	99				

PROGNOSIS AFTER OPERATION FOR CLEFT PALATE.

The prognosis after operation depends on many factors, the first being the age at which the operation is done—that is, whether done before or after speech is developed. It can be said that where the operation is done during infancy, and *where a functional palatopharyngeal valve has been formed*, speech will develop along normal lines without any training. It should be the aim of every surgeon who is dealing with these cases to put the speech trainer out of work, and the only way to do this is by operation before faulty speech habits are formed. In an earlier part of this paper mention was made of the imaginary attempts of the sufferer from cleft palate to overcome his lack of a palatopharyngeal sphincter, and the importance of operation at this stage, before the development of articulatory gymnastics, cannot be overstressed. Cleft palate is a deformity which should be treated as a surgical emergency, and should be submitted to operation as soon as the health of the child and the skill of the surgeon will allow. Operation is often extremely difficult, but rarely dangerous, and requires patience, courage, and skill with sometimes but little reward. Nevertheless such deterrents should not weigh in the attempt to obtain better results in this most distressing complaint.

When the sufferer from cleft palate has reached that stage at which speech has been acquired, the whole question of prognosis takes on an entirely different aspect, and within limits this aspect is not materially altered by the anatomical defect present. Apart from the surgical difficulties, the main factor to be taken into account is the character of the speech acquired. As was mentioned previously, cleft-palate speakers divide themselves into three rough groups, and it stands to reason that in given anatomical circumstances operation on these three groups will produce widely different results. If all are provided with a properly functional mechanism, although the result of the operation is the same, the speech will deteriorate in order from the first to the third group. A great deal of literature has been produced on the speech results of operation for cleft palate, but in none that has come under my notice is this, the most important feature, noted, and it would seem to be obvious that no record of results after the age of speech is of the slightest use unless the speech before operation is taken into account. It is worth while examining these three groups in detail, *assuming that as the result of surgical treatment each has been provided with a functional palatal mechanism*.

Group 1.—In the first group the consonants are formed in the correct way but with nasal escape. In every such case it is almost certain that normal speech will be restored after operation, and this may be done without any training whatever, the only stigma likely to remain for some time being the 'nasal grimaces' which are so characteristic of the habit acquired while attempting to prevent nasal escape. In the reasonably intelligent patient a few lessons demonstrating the use of the palatopharyngeal valve may be necessary, and this may be all that is required. Two of my cases (*Cases 18 and 80*) fell into this group and are recorded above.

Group 2.—In the second group is included the greater part of the cases in the average surgical practice. The patients have acquired faulty speech

technique which may render them almost unintelligible to the uninitiated listener. They are the quintessence of joy to the speech trainer, since they provide his livelihood for many a long day. The habits of a lifetime have to be unlearned and new habits acquired, a feat which becomes increasingly difficult as age advances. Of all the cases of cleft palate these are undoubtedly the worst to manage, because, in spite of an operation which should be regarded as highly successful in its anatomical and physiological result, speech may improve but little. Acuity of hearing, a certain amount of intelligence, and, above all, ambition, are necessary for complete success, and up to the present these factors are entirely beyond the control of surgery. It is sometimes marvellous to behold the good influence of an approaching courtship or marriage, and sometimes also to observe the return of carelessness after the desires are attained. Amongst my greatest disillusionments in cleft-palate surgery was the case of a young woman who belonged to this group (*Case 5*) and upon whom the operation of pharyngoplasty with repair of the palate was performed. The operation resulted in a perfect palatopharyngeal mechanism which complied with all the tests, and for many weeks I spent much time in correcting her bad habits, until in the end she was sound perfect. At this time she was engaged to be married, and, marriage ultimately taking place, I did not see her till some months after, when, happening to meet her in the street, I was amazed to find that she had become one of the worst cleft-palate speakers I had ever heard.

It must surely, then, be realized that while surgery is able to provide a perfect speech mechanism, and speech training in the suitable case can complete the cure, there is no operation which will make the patient use the mechanism and the training in the ordinary duties of life. This case is amongst my best results, yet remains amongst the worst speakers, and it is hardly fair to include it in the surgical failures, but it certainly supports the plea put forward to regard the cleft palate of infancy as an emergency.

In this and the next group there is a certain proportion of the patients who are quite untrainable from lack of ambition, self-consciousness, or sheer crass stupidity, and it is felt that it is equally unfair to attribute this to any operative procedure, since they may possess the mechanism, but for the reasons mentioned above are quite incapable of using it. There is much to be said for the method of Ernst, who, in dealing with cases after the speech has been acquired, first of all fits them with an obturator and attempts to train them in speech; if they are trainable, he proceeds to operate.

Group 3.—The third group contains all those who are incapable of forming consonants at all, and who are consequently unintelligible. The glottic stop is usually freely used and serves to separate the vowels from one another, and in this way a form of speech is acquired. It is wrong to assume that such cases are lacking in intelligence, acuity of hearing, or any other abstract quality, as the following will show. A boy of 6 years was brought to me with speech of this type (*Case 22*). He had a cleft of the soft palate which also involved about half the hard palate. Speech was quite unintelligible. Pharyngoplasty was performed, and fourteen days later the palate

was repaired by the Langenbeek-Fergusson method. Within three weeks of the second operation he was able to use his palatopharyngeal valve and could blow soap bubbles. Within three months all his consonants, the result of speech training, were perfect. He now speaks perfectly and without the slightest stigma of cleft palate, although he possesses a broad Tyneside accent. This boy has been lucky in that he has never acquired thoroughly bad speech habits, and the only trick he has had to unlearn is the glottic stop. He is an intelligent little lad and gives the lie to those who suggest that all patients in this group of speech are all mentally deficient. That some may be mentally dull is true, but I have the feeling that I would rather tackle some cases in *Group 3* than a great many of those in the previous group.

SPEECH TRAINING.

Most of the literature dealing with cleft palate finishes in pious vein with the suggestion that an essential feature in treatment consists of training after operation, and there the matter ends. My experience of cleft palate has taught me that if successful results are to be obtained there must be close co-operation between the operator and the trainer. One cannot get on without the other, each working in a little watertight compartment, and if they are to co-operate with useful effect it is necessary that the trainer should understand the essential surgical and physiological requirements of the operative treatment, and for this purpose there is no better educational exercise than the attendance at a few operations.

Speech training is not only something to be used after operation where the result is obviously bad. It can be used to some purpose before operation by an intelligent mother where a child with cleft palate is first beginning to talk, and if she is successful she will assure that the operation will restore normal speech. During this time of life it is most important that the child should be taught correct habits in that the tongue, lips, and teeth should be placed in their correct positions for the formation of any given sound. Where a child is obliged to await operation it is my custom to instruct the mother either not to encourage the child to talk at all, or to see that if it does talk the sounds are formed in as perfect a manner as possible.

But there is no doubt that it is after operation that speech training finds its greatest sphere of usefulness, and its method of application depends on whether the child has been operated upon before or after the talking age.

If the operation has been completed before the child talks, the training as such rests, as it does in most of the population, in the hands of the mother, and, apart altogether from cleft palate, the mothers of this world are the greatest speech trainers at all times. But much can be done to help at this stage, and the first essential is to make the child 'valve conscious' by means of any form of blowing exercise which for its successful execution requires closure of the palatopharyngeal valve. Trumpets, squeakers, soap bubbles, and balloons are popular toys with children and can be made to exercise an immense influence on the subsequent course of speech. It has been my experience to observe on many occasions the improvement which takes place when blowing exercises are religiously carried out, and many children in

whom the baby talk is obviously cleft-palate in type are restored to normal speech within a few months. It was very interesting to observe one child who improved in speech until everything was perfect with the exception of some of the double consonants—for instance, the *s* as in 'swim'. In the course of a few months even this defect was completely corrected—a great tribute to the value of operation at or before the age of experimental speech.

Where the operation has been completed after speech has commenced, the training becomes a much more complicated process, and resolves itself into the correction of faults as well as instruction in the proper method of forming sounds. However, it is advantageous to discuss this question in relation to the various groups described earlier on. To recapitulate: *Group 1* consists of those whose only fault is nasal escape; *Group 2* have learnt all the articulatory faults; *Group 3* are quite unable to form any consonants but use vowels only, with or without the glottic stop.

Group 1 speakers have only to be taught the use of the palatopharyngeal valve, and gain very material help from blowing exercises or from learning to play some wind instrument. Sometimes they have difficulty at first with blowing, and can often be helped by being instructed to snort several times and then to blow, keeping the soft palate in the same position as during the snort. It is not often that failure follows a demonstration of this kind. There is only a short jump between the successful blowing exercises and the perfect formation of the consonants without nasal escape, since, where a defect is found to exist, snorting followed by a repetition of the consonant is usually effective, and has proved an immensely good method in many of my cases. This is really all that is required with the *Group 1* patient, and my experience has been that perfect speech follows as a natural sequel. Bad habits can be controlled from time to time by the use of a small mirror placed horizontally and glass side up beneath the nostrils, when any nasal escape is signified by steaming of the glass.

Group 2 cases present the greatest problem in that their faults have to be remedied and replaced by normal methods. There are three main faults in this type of speech: (1) Nasal escape; (2) Complete absence of a consonant and its replacement by the glottic stop; and (3) Formation of the sound in the wrong position. Nasal escape can be corrected by the method outlined above for *Group 1* cases. Absence of the consonant and its replacement by the glottic stop is one of the commonest faults and is very difficult to eradicate, since, as a habit, it is usually well ingrained. The consonants affected by this method of speech are mostly the *t*, *d*, *k*, *g* (hard), and any words containing these letters, at any rate in their middle or at the end, are simply slurred over. In most cases the tongue is placed within reach of the correct position, but instead of interrupting the air-stream in the mouth it is interrupted in the glottis, an illustration of the misuse of the glottic stop. The word 'better' thus becomes 'be/er', 'adder' becomes 'a/er', 'sparkle' becomes 'spar/le', and so forth, the stroke in each case representing the glottic stop. Particularly obnoxious is this habit in words terminating in these consonants, as in 'but', which becomes 'bu/'.

In restoring the *k*, *g*, *t*, and *d*, it is best to start with the voiceless sounds

and demonstrate the position in which it is necessary to place the tongue in relation to the hard palate and attempt to form the explosive effort as 'kuh', 'tuh', rather than uniting consonants with a vowel as in 'kay' or 'tee'. Sometimes nipping the nostrils to prevent nasal escape assists materially in this. The keen patient rapidly acquires this feat and one may then proceed to introduce vowel sounds, giving as exercise 'kar, kay, key, ko, koo', and also 'ack, ache, eke, ike, oak, ook', etc. From this the voiced consonants are then introduced, and finally double consonants.

Formation of the consonant in the incorrect position is best illustrated by the cleft-palate *s*, where the sound is made somewhere in the pharynx, the exact site of which I have never been able to locate accurately. This sound can be made with the mouth wide open, a feat impossible for the normal speaker, and although I have learned to mimic the cleft-palate speech with a considerable amount of success, I have never been able to reproduce the cleft-palate *s*. The correction of the faulty *s* is not really difficult, and can be done by instructing the patient to place the tongue in position as though about to say *t*, but, instead of making the explosive effort, to allow the air to escape between the tip of the tongue and the anterior end of the hard palate immediately behind the alveolus, the trainer meanwhile nipping the nose if necessary. One of my cases after learning the correct method was unable after the lapse of about a fortnight to repeat for me the cleft-palate *s* which had been a life-long habit. Having learnt the correct *s*, the next procedure is to practise this sound in association with various vowels, the latter respectively preceeding and succeeding the consonant. Finally comes the double consonant, the *sk, sl, sm*, etc., and these sometimes offer at first some considerable difficulty, but this can be overcome by taking each consonant separately and then conjoined.

In only one of my cases can I claim that perfect speech in *all circumstances* has been secured as the combined result of operation and speech training, and this patient belonged to *Group 3* in which no consonants and no fault beyond the glottic stop had been acquired; before operation he was quite unintelligible.

Although speech training will improve the patient beyond all recognition, it will never produce perfect speech, since the acquisition of this depends entirely on the development of a habit and represents a tremendous uphill fight.

Group 3 speakers possess only the vowels, which are used with or without the glottic stop. This group includes a curious mixture of cases in which speech sense and mental dullness are encountered in varying proportions. Mention has been made previously of a patient in this group who within three months of operation was sound perfect and ultimately developed into an habitual normal speaker (*Case 22*). Unfortunately not all in this group display such speech sense, as it is quite a common experience to meet with the type who is quite unable to imitate even the simplest sounds, and it is very difficult to be sure whether this is the result of a failed operation or just stupidity. One thing is quite certain—namely, that the type of cleft present has no real influence on which particular group of speech the patient will adopt.

PHARYNGOPLASTY AS A SECONDARY OPERATION.

There is an enormous number of cases in this country in which the palate has been repaired by one method or another, but in which no functional valve has been formed. Every speech-training clinic is filled with such patients, and much valuable time and energy is wasted in attempting to restore normal speech to those who do not possess the necessary mechanism—an effort which can only be compared to attempting to obtain music from a broken flute. Yet it is possible in some of these cases to restore the palatopharyngeal valve and thus ensure that at any rate the flute is repaired in an efficient manner, with consequent improvement in speech and the chance that the patient may, should he or she so desire, acquire perfect speech. Unfortunately the opportunities of carrying out work of this kind are few and far between, since patients do not readily submit to further operation when they have tasted failure from a previous operation done for the same condition.

However, I have had the opportunity of performing pharyngoplasty on ten occasions where the palate had previously been successfully repaired by others and the speech result was bad. Although a considerable degree of thought has been devoted to this aspect of cleft-palate surgery, the amount of experience afforded has not allowed of any firm conclusions with regard to technique in the treatment of the palate itself. One fact stands out quite clearly, however—namely, that pharyngoplasty is capable of restoring a valvular mechanism, and in the right type of case causes immense improvement in speech. Of the 10 cases mentioned, only 2 possessed any mobility in the soft palate, all the remainder being firmly fixed as a result of previous interference. Of these 10 cases, 3 live in distant parts of the country and have consequently not been submitted to personal observation since operation, and 1 lives nearer but has not presented himself for review. Of the remaining 6, 4 present evidence of palatopharyngeal valves and 2 are complete failures.

Pharyngoplasty after repair of the palate presents no serious difficulties, as the soft palate can easily be held forward by means of a small hook or an aneurysm needle and the operation can be completed as in the unrepaired case. All attempts to secure mobility in the soft palate have in my hands failed, although I believe that by division of the hamular processes and freeing the posterior end of the hard palate a little more has been secured in length. Further length in the palate has also been obtained by paring the free edges of the posterior pillars of the fauces for about $\frac{1}{2}$ in. and then suturing in the mid-line. This brings the posterior pillars into great prominence, and the uvula projects forwards in a nipple-like fashion, while any remaining tonsillar tissue points in a more forward direction.

The 6 cases in which secondary operations have been done all presented themselves with immobile soft palates, so that the problem of restoring the palatopharyngeal valve was materially increased. These cases are worth studying in detail.

Case 25 was that of a girl aged 9 years. She had had a wide median cleft of the soft and hard palates almost reaching the alveolar margin. Several operations of the classical type done previously had resulted in

complete fixation of the soft palate and there was a large hole in the hard. Speech was of the type noted as *Group 2*—that is, with the development of all the faults. Pharyngoplasty was done in June, 1931, and as a result speech was much improved. At a second operation the mucoperiosteum of the hard palate was stripped from the bone in the form of two flaps based on the posterior palatine artery, and the hamular processes were destroyed, the operation being completed by suture in the middle line. The improvement in speech was immediate. This child can now snort strongly; her speech is still of the cleft-palate type, but in my presence she was able to make the principal consonants without any nasal escape whatever. The s was of the cleft-palate type and could be made with the mouth wide open, but after a few minutes' demonstration she was able to make it the correct way and without nasal escape. This child has been undergoing training, but not under my supervision.

Case 32 was a girl aged 14 years who had a median cleft of the soft and hard palates repaired at the ages of 2 and 2½ years. After the first operation the palate broke down completely, but held after the second attempt. The soft palate was completely immobile, but the speech was very good, the only defective sounds, apart from the gross nasal escape, being the *k* and *g*. At operation in May, 1931, pharyngoplasty was done by the usual method, and it was noted that immediately afterwards speech improved immensely; but unfortunately this was not completely maintained owing to some of the sutures giving way. At a second operation in September, 1932, a further attempt was made to increase the pharyngeal narrowing of the first operation, and the posterior edges of the soft palate and posterior faucial pillars were pared and sutured in the mid-line. Unfortunately all the sutures gave way and there was no further improvement. As a result of all this operating the patient is able to snort, but only weakly; her speech is very much better, but she is still unable to effect fully competent pharyngeal closure.

Case 39 was a boy aged 7 years. He had had a bilateral complete cleft of the hard palate with a double hare-lip operated on within three years of birth. When I first saw him his speech was quite unintelligible. The palate was rigid and lay far from the pharyngeal wall. At operation in 1931 the mucoperiosteum of the hard palate was stripped up in the form of two flaps based on the posterior palatine artery and the hamular processes were destroyed. A large hole was left at the anterior end of the hard palate and this was later closed by an obturator. A very satisfactory pharyngoplasty was done at the same time. There was some immediate improvement in speech, but as it was not felt that the maximum possible surgical closure had been effected, a second operation was decided upon and carried out early in 1932, when the pharyngoplasty was further tightened and the uvula and the posterior edges of the soft palate and posterior pillars of the fauces were pared and sutured in the mid-line. This boy has been under constant observation since and has been trained by Mr. T. W. Moles. Although he was an unintelligible *Group 2* speaker at first, he is now able to obtain every sound perfectly. He can snort very strongly and can inflate a rubber balloon. Training has been discontinued. His father writes, "Mr. Moles is of the opinion that I can now complete the training myself as G— can get all

the sounds *when he likes* and it is now mainly a matter of continued exercise." The italics are the father's.

Case 72 was aged 5 years at the time of operation. He had a complete unilateral cleft of the palate with a hare-lip which had been operated upon within two years of birth. On first being seen, the cleft was completely closed but the soft palate was immobile. At operation in June, 1928, pharyngoplasty was done. The boy was not seen for over four years, when it was found that he had a perfectly competent valve which enabled him to inflate a balloon in my presence. Speech was still of the cleft-palate type in *Group 2*, but no training of any sort had been given. yet in my presenece he could be made to say many consonants correctly.

Case 33 was aged 12 years. He had had a median cleft of the soft and hard palates which had been repaired, resulting in a hopelessly immobile soft palate with much scarring. The nasopharynx was of enormous proportions. Speech was in *Group 3* and quite unintelligible. Pharyngoplasty was carried out but made no improvement, the boy being unable to imitate any sound whatever. It should have been recognized before operation that he was hopelessly dull mentally and not worth any waste of time or effort.

Case 46 was a woman aged 24 years with a previously repaired bilateral cleft. The lip was extremely bad and an enormous hole existed in the hard palate. The soft palate was immobile and short. Speech consisted of vowel sounds with the glottic stop and an occasional consonant. Pharyngoplasty was done in April, 1932, and although it appeared to be a very satisfactory surgical result there is no evidence of valve formation nor is speech improved.

Comment.—In the cases outlined above 4 out of the 6 have now palato-pharyngeal valves, although in *Case 32* the valve is not fully competent, a fact which is supported by the weakness of the snort. Three of the cases present no doubt as to the efficiency of the valve, and this in spite of an immobile palate, and it is interesting to observe how the ridge of a pharyngoplasty is able to compensate, at any rate in the simpler exercises, for this loss. The opportunity of operating upon and following the subsequent progress of a case with a freely mobile palate is awaited with interest.

REFERENCES.

¹ *Jour. of Anat.*, 1930, lxx, Pt. 1, October.

² *Brit. Jour. Surg.*, 1928, xvi, 127.

³ PAGET, SIR RICHARD, Bart., *Human Speech*, 1930. London: Kegan Paul, Trench, Trubner, & Co. Ltd.

*SHORT NOTES OF
RARE OR OBSCURE CASES*

**GRANULOMA OF INTESTINE. STENOSIS OF
ILEOCÆCAL VALVE.**

BY H. W. L. MOLESWORTH, FOLKESTONE.

A WOMAN aged 30 had had an appendicectomy done in 1921 by another surgeon. At that time she complained of vomiting and slight right-sided abdominal pain; these symptoms were relieved by the operation, and she remained perfectly well until July 4, 1932, when she had a short attack of colicky abdominal pain associated with nausea, but without vomiting or constipation. This lasted for a few days and she got completely well.

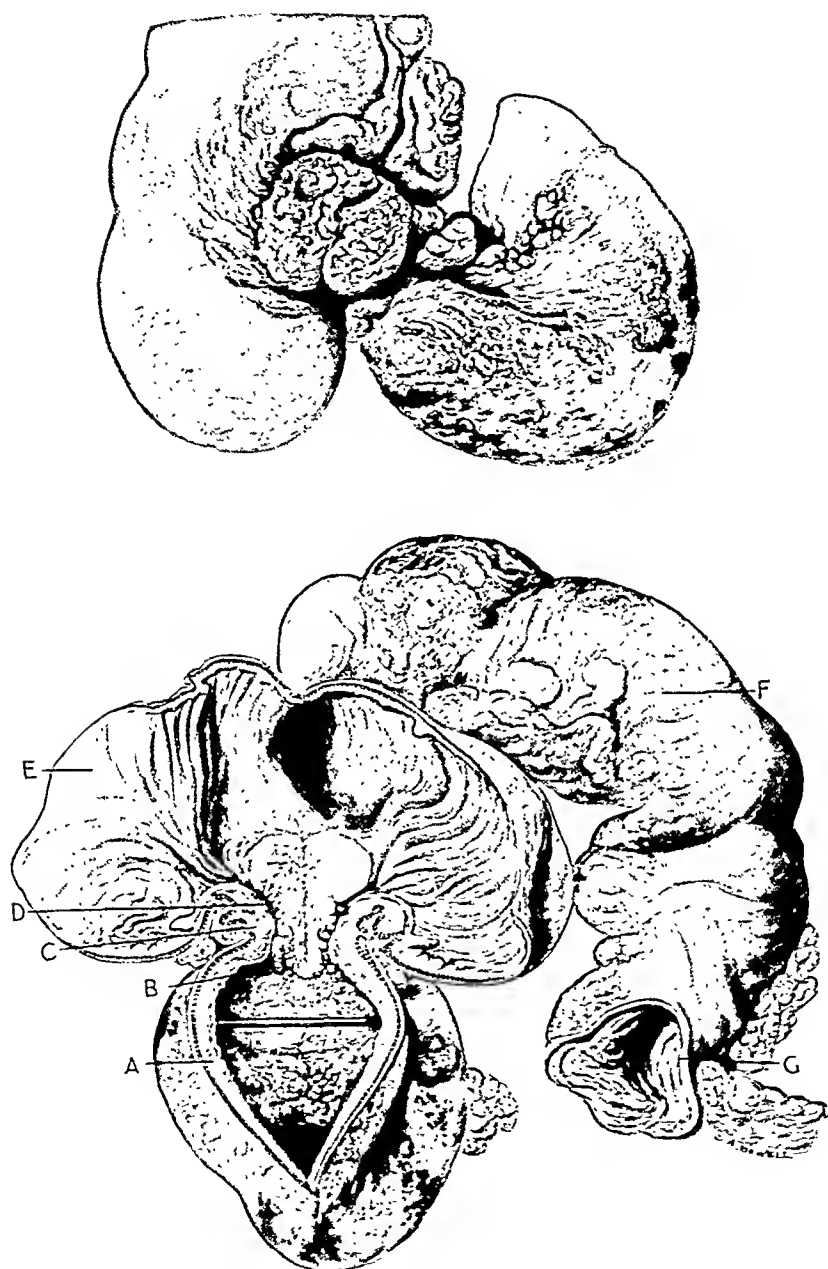
Towards the end of July she underwent an operation for hallux valgus; she was in hospital in London and during this time complained of no abdominal symptoms. On Aug. 18 she complained of colicky abdominal pain and nausea. Up to Aug. 23 the bowels acted with regularity. On Aug. 24 pain was much more severe. The patient was seen by me that evening and was then in a condition of obstruction to the small intestine.

A laparotomy was done the same evening. The small gut was distended and hypertrophied and there was a swelling in the region of the ileocæcal valve which somewhat resembled that found in congenital pyloric stenosis. The terminal few inches of the mesentery of the intestine were markedly thickened and indurated, as was the terminal portion of the small gut. An anastomosis was made between the small intestine and the transverse colon. At this stage there were no adhesions connected with the small intestine. Those that exist on the specimen subsequently removed have occurred since the first operation.

On Sept. 9 the abdomen was re-opened, and removal of the ileum, ascending colon, and about one-third of the transverse colon carried out. The operation was rendered difficult by numerous adhesions between the ascending colon and the small intestine. The patient has made a good recovery and is well at the time of writing.

The drawings by Mr. Sewell (*Figs. 259, 260*) show the condition found, and Mr. Lawrence has kindly permitted me to make use of his description of the specimen, which is in the Museum of the Royal College of Surgeons:—

"Stenosis of ileocæcal valve: The terminal 6 in. of the ileum, with the cæcum, ascending colon, and first part of the transverse colon, which were removed by operation. An incision has been made in the free border of the ileum and cæcum, displaying a tight stricture of the ileocæcal valve, admitting only of a narrow probe. The stricture is about $\frac{3}{4}$ in. in length and the wall of the intestine bounding it is thickened to the extent of $\frac{1}{2}$ in., the thickening



FIGS. 259, 260.—Specimen removed at operation, with the ileum and cecum shown open below. A, Dilated and hypertrophied ileum; B, Polypous mucosa; C, Organizing granulation tissue; D, Stricture; E, Cæcum; F, Ascending colon; G, Transverse colon.

being due mainly to a fibrous-looking increase of the submucous layer; the thickening is continued for 2 in. along the adjoining part of the ileum. The lining of the stenosed segment is the seat of small polypoid excrescences. Partly covering the stenosed portion on the external surface of the bowel is a mass of dense fibro-fatty tissue measuring $1\frac{1}{2}$ in. in width. The vermiform appendix is absent. The terminal part of the ileum is dilated and hypertrophied; its lining is irregularly thickened, and exhibits small papillary projections in places. Its surface is covered with dense adhesions, a delicate membranous adhesion also covers the cæcum and ascending colon; the mucous membrane of the latter is free from ulceration. No enlarged glands are present in the specimen.

"Microscopical examination (*Fig. 261*): The mucous membrane is ulcerated on both sides of the stenosis. On the surface there is a layer of coagulated pus, beneath which is a broad zone of granulation tissue.



FIG. 261.—Microscopical section of the wall of the ileum. ($\times 25$.)

A thick formation of fibrous tissue replaces the submucous layer, and the two muscular coats are hypertrophied and separated by a similar formation. Both fibrous and muscular layers contain numerous foci of leucocytes, mostly arranged around vessels. At the margin of the ulcer the mucous membrane is raised in the form of small polypi or papillomata; these are covered with cylindrical epithelium, among which are numerous goblet cells. There is no evidence of carcinoma; no downgrowth of epithelium at the base or margin of the ulcer.

"It is probable that the stenosis of the ilcoæcal valve was a sequela of the appendicitis and appendicectomy."

This condition would appear to be identical with that described by Mock,¹ and more recently by Erdmann and Burt,² whose articles contain a list of references to the literature.

REFERENCES.

- ¹ Mock, *Surg. Gynecol. and Obst.*, 1931, lli, 672.
- ² ERDMANN and BURT, *Ibid.*, 1933, lvii, 71.

A SOLITARY PLASMOCYTOMA OF THE FEMUR.

BY CYRIL POLSON AND N. SHIRES,

FROM THE DEPARTMENTS OF PATHOLOGY AND BACTERIOLOGY OF THE UNIVERSITY
AND ST. JAMES'S HOSPITAL, LEEDS.

DURING 1932 two publications, those of Stewart and Taylor,¹ and Harding and Kimball,² concerned solitary plasmocytoma of bone. Each reviewed the literature, giving indication of the rarity of this neoplasm. The former authors described two original cases, one a plasmocytoma of the humerus and one which probably arose in the maxilla, while the latter authors described a plasmocytoma of the femur.

In the case described by Rogers,³ the patient, who had a solitary plasmocytoma of the right femur, came under observation on account of pathological fracture of the bone following a slight accident. Amputation of the leg resulted in his complete recovery.

The present case resembles that of Harding and Kimball, since it was a growth of the femur at first considered a secondary deposit of carcinoma.

HISTORY.—The patient was a male aged 72 years who was admitted to St. James's Hospital on Sept. 5, 1928, on account of chronic gastritis and hæmorrhoids. He was re-admitted on Oct. 4, 1932, because he had slipped on the stairs at his home and fractured his left femur. During the previous fortnight he had had pain in this leg and used a stick when walking. He also complained of irregularity of the bowels and difficulty in micturition.

ON ADMISSION.—The patient had a fracture in the middle third of the left femur, and after radiological examination Dr. Wall reported that this was a pathological fracture (*Fig. 262*). The leg was placed on a splint. No examination of the urine for the detection of Benec-Jones protein was made. Twelve days after admission (Oct. 16) his abdomen became severely distended, but no tenderness or rigidity was apparent. He vomited, and an enema produced no result. A diagnosis of intestinal obstruction, probably due to a carcinoma of the bowel, was made.

OPERATION.—The abdomen was opened under local anæsthesia. The large intestine was grossly distended and a transverse colotomy was performed.



FIG. 262.—Radiogram of left femur showing evidence of rarefaction but no expansion of the bone in the affected area.

The circumstances precluded any extensive examination of the peritoneal cavity. Although some improvement followed, the patient died on Oct. 20.

POST-MORTEM FINDINGS.—

At autopsy the body was that of a well-nourished elderly man. His left leg was shorter by 3 in. than the right and externally rotated. No external abnormality of the left thigh was observed nor was any tumour palpated, but upon manipulation abnormal mobility was present at the middle of the femur. Examination of the bone showed that there was an ununited fracture in its middle third with over-riding of the fragments. In the process of clearing the bone of its muscle sheath, yellowish growth, infiltrated with blood, was encountered on the outer aspect of the bone and adjacent to it in the region of the fracture. The mass of growth

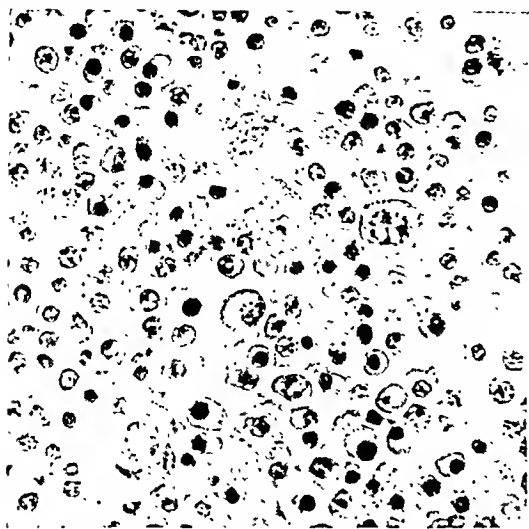


FIG. 263.—Microphotograph showing types of cell. A large 'pre-plasmacyte' can be seen in the right upper quadrant. ($\times 100$.)

was small and was less than 3 in. in diameter. Little or no expansion of the bone had occurred, but there was destruction of about 1 in. of the shaft in the region of the fracture. Rarefaction of the bone was present for a distance of about 2 in. above and 1 in. below the fracture, and here the medullary cavity was filled with growth of a soft consistency and greyish colour. Death was due to acute perforation of a chronic duodenal ulcer situated on the anterior wall. There was but little peritonitis or escape of fluid. Except for one or two small adenomata of the prostate no other neoplasm was found. Early bronchopneumonia was present as a terminal condition.

MICROSCOPICAL FINDINGS.—

The femoral tumour was subjected to histological examination and proved a plasmocytoma. Two types of cell were abundant.

First an ovoid or pyriform cell having an eccentric nucleus containing plentiful

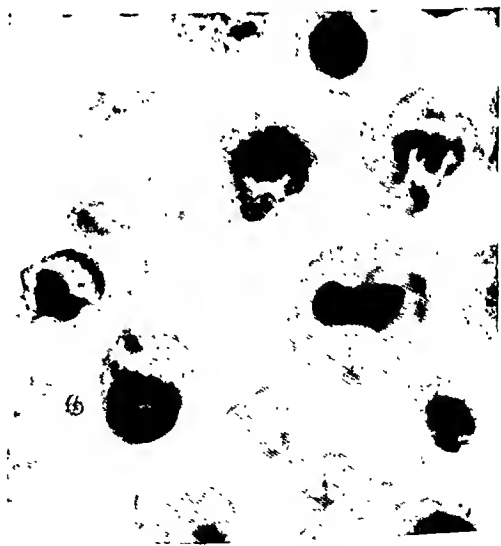


FIG. 264.—Microphotograph showing plasma cells, one containing a mitotic figure. ($\times 1200$.)

chromatin frequently arranged in 'cart-wheel' pattern (*Fig. 263*). The protoplasm yielded the characteristic reaction with Pappenheim's stain, and a perinuclear halo was observed in a small proportion of these cells. They were plasma cells, but of rather larger size than those usually seen in chronic inflammatory foci. The second type of cell was even larger and possessed a large nucleus, centrally situated (*Fig. 263*). The protoplasm yielded the Pappenheim reaction, and these cells are considered to be immature plasma cells.

Mitosis was frequent in certain areas of the growth (*Fig. 264*). A count of approximately 7500 cells in 100 adjacent fields viewed with the $\frac{1}{2}$ objective showed that mitosis occurred in 1 per 200 cells. Several phases of mitosis were observed, and the cells concerned yielded the Pappenheim reaction, although their protoplasm was a little paler than that of other plasma cells. Multinucleated cells and lymphocytes were scarce and distributed through the growth. There was a sparse collagenous stroma in which were a number of blood-vessels, most being well formed. As in Stewart and Taylor's cases, the stroma fibrils were, in some areas, arranged in parallel with columns of plasma cells between. The areas of hæmorrhage were ascribed to the violence of fracture.

COMMENTARY.

It is scarcely a matter for surprise that the diagnosis of the femoral lesion was not made during life. Moreover, even in the presence of much less evidence calculated to mislead, it is not unlikely that a plasmocytoma may be mistaken for a secondary deposit of carcinoma. It is imperative that these two conditions should be differentiated, since the local malignancy of solitary plasmocytoma has been demonstrated and since local treatment may result in a complete cure. The necessity of a biopsy before proceeding to radical surgery has been insisted upon by Stewart and Taylor, and the present case bears out this conclusion in an unmistakable fashion. Harding and Kimball comment upon the fact that plasmocytoma of the appendicular skeleton invariably causes pathological fracture. We therefore insist that the reasonable procedure in all cases of pathological fracture is an open examination of the lesion and the removal of tissue for biopsy. Should it be a secondary deposit of carcinoma, then the prognosis is determined, whereas if it is a solitary plasmocytoma, much may be done, not only to save the patient's life, but also to restore him or her to health and activity.

Since a complete examination of the skeleton was not made in this case, the tumour was not proved to be a solitary plasmocytoma, although this was almost certain. The essential histological features of the growth were in accord with those described by other authors, but there was evidence of unusual activity, since large 'pre-plasmocytes' were plentiful and numerous mitotic figures were seen.

We are indebted to Dr. J. Wall for permission to reproduce the radiogram.

REFERENCES.

- ¹ STEWART, M. J., and TAYLOR, A. L., *Jour. Pathol. and Bacteriol.*, 1932, xxxv, 541.
- ² HARDING, W. G., and KIMBALL, T. S., *Amer. Jour. of Cancer*, 1932, xvi, 1184.
- ³ ROGERS, H., *Brit. Jour. Surg.*, 1930, xvii, 518.

REVIEWS AND NOTICES OF BOOKS.

Poliomyelitis. A Survey made possible by a Grant from the International Committee for the Study of Infantile Paralysis organized by Jeremiah Milbank. Large 8vo. Pp. 562 + xxii, with 25 plates. 1932. Baltimore: The Williams & Wilkins Company. (London: Baillière, Tindall & Cox.) 35s.

This volume represents four years' work, carried out by grants from the Fund, of forty-four practical workers at a cost of 280,000 dollars. It contains a comprehensive survey of poliomyelitis so far as the history, etiology, symptoms, pathology, and epidemiology are concerned. But it omits treatment entirely except prophylaxis and the treatment of the acute attack by serum and other similar measures. The references are very extensive, occupying 43 pages, and as there is no indication to show which portions of the work summarized have been carried out by the investigators who have received grants from the Committee, it is difficult to form any opinion as to whether the results are really commensurate with the amount of work done and the cost incurred.

The chapter on epidemiology is very full and of much interest. It is noticeable that, whereas in Sweden the case rate per 100,000 has risen above 10 on several occasions, with a peak of 70, and that the rates have also been high in the United States and New Zealand, in England this rate has never been as high as 5. In the opinion of the Committee there has been no proof that the administration of convalescent serum in any way alters the severity of the attack or the prognosis as to the severity of the resulting paralysis. This does not appear to agree with observations in other countries.

The book will be consulted by those who are interested in research work upon this disease, but is of no real value to a clinician.

The Principles of Treatment of Muscles and Joints by Graduated Muscular Contractions. By MORTON SMART, C.V.O., D.S.O., M.D., Ch.B. (Edin.), Late Medical Officer in Charge of the Electrotherapeutic Department, Hospital for Sick Children, Great Ormond Street. Large 8vo. Pp. 217 + xvi. 1933. London: Humphrey Milford. 15s. net.

MORTON SMART'S method of treatment by graduated muscular contractions, produced by stimulation with a faradic coil of special pattern, is now very widely known and practised. This personal and authoritative description of his methods is therefore very valuable, and it is essential that it should be read by all those who practise physical treatment. In the earlier chapters there is a general account of the physiology of muscular action and the pathology of some of the conditions treated. Following this, manipulation of a shoulder-joint for adhesions and after-treatment are described and then a general account of muscle treatment is given. No doubt there will be many readers who do not agree entirely either with the author's methods or with his opinions. For example, to force abduction and rotation of the shoulder directly (without traction) may be a dangerous form of manipulation, risking fracture if there are firm adhesions. Morton Smart believes that faradic stimulation of muscles will increase their strength and volume; many will hold that voluntary action will do so more successfully. And many will feel that to give as large a number as 1800 contractions to a muscle at each sitting may well mean overworking it if it has been disused. In general also we may believe that the faradic treatment of muscles should be largely a preparation for the use of active movements and exercises, a side of the work which is not stressed in this volume.

However, these criticisms do not detract from the value of the book in its description of methods and in the final chapter of details of the apparatus used.

Treatment of Fractures in General Practice. By W. H. OGILVIE, M.D., M.Ch., F.R.C.S. Fcap 8vo. Vol. I, Pp. 108 + viii; Vol. II, 109-180. Illustrated. 1932. London: John Bale, Sons & Danielsson Ltd. 2s. 6d. each.

THIS little book is an excellent example of how much can be compressed into a small space. It contains an outline of the anatomy, including development of epiphysal nuclei, the mode of causation, pathology of repair, and treatment of fractures, all put in a form which can very easily be consulted by a general practitioner.

Naturally in the case of fracture treatment the methods are those of the author and would not necessarily be accepted in detail by everyone, but there is little to be found fault with. The principles are sound throughout and methods described with sufficient detail and with simple but adequate illustrations. Mr. Ogilvie has introduced the name of "cancellous fracture" in injuries of such bones as the os calcis and vertebral bodies instead of the term "crush fracture" which is more often applied. One omission is that of any description of the use of the X-ray operating table in the setting of fractures. The illustration of a Colles's fracture after reduction shows the wrist in an extended position, a method which would be generally condemned, and under the treatment of delayed union the use of parathormone is advised. Considering that the latter is now known to cause decalcification of the bones its use for this purpose should not be advocated in a modern text-book.

The Injured Workman. By G. F. WALKER, M.D., M.R.C.P., with the collaboration of J. HARVEY ROBSON, Barrister-at-Law (Law); R. E. JOWETT, M.D., Ch.B., D.L.O. (Ear, Nose, and Throat Surgery); STANLEY RITSON, M.B., M.S., B.Sc., F.R.C.S. (General Surgery); JOHN FOSTER, M.A., M.B., F.R.C.S., D.O.M.S. (Ophthalmic Surgery). Foreword by W. H. MAXWELL TELLING, M.D., B.S., F.R.C.P., Professor of Forensic Medicine, University of Leeds. Crown 8vo. Pp. 190 + xx. 1933. Bristol: John Wright & Sons Ltd. 6s. net.

THIS work deals with the legal aspects of the late results of an accident to a workman, and by 'accident' is here meant a single blow, stress, or injury. An impressive foreword is contributed by Dr. W. H. Maxwell Telling, who rightly points out the great difficulty that exists in finding 'light work' for the injured workman during the stage of transition from partial to complete recovery—a period when the most important factor in therapeutics is to get him back to 'something' in the way of work as soon as possible.

In the first chapter the essential features of the Workmen's Compensation Acts are well epitomized, but in other parts of the volume it is not sufficiently emphasized that compensation is awarded not because an injury has been received as the result of an accident arising out of and in the course of employment, but because that injury has resulted in an actual loss of earning capacity. The section on "Rusting and Brooding" in Chapter II may be especially commended for its philosophic thought and its sound common sense.

The special injuries of the various regions of the body are considered in detail, but here a sense of proportion seems to have been wanting. Thus, of a total of 128 pages devoted to the whole of the body no fewer than 50 are concerned with the eye—injuries of which form a relatively uncommon cause for compensation—while the infinitely more important injuries to the fingers and hands are very summarily dismissed.

The Cure of Hæmorrhoids, Varicose Veins and Ulceration, and Allied Conditions: by Modern Methods and Bandaging. By STUART McAUSLAND, B.A. (Lond.), M.D., Ch.B. (Liv.). M.R.C.S., L.R.C.P., Hon. Physician, Hahnemann Hospital, Liverpool, etc. Large 8vo. Pp. 63. Illustrated. 1933. London: John Bale, Sons and Danielsson. 3s. 6d. net.

THIS is a small handbook on the injection treatment of varices and hæmorrhoids. It is well illustrated and gives some practical suggestions as to the methods, drugs, and instruments used. It will serve as a cheap and useful guide to the general practitioner.

The Diagnosis and Treatment of Postural Defects. By WINTHROP MORGAN PHELPS, B.S., M.D., M.A., F.A.C.S., Professor of Orthopaedic Surgery, Yale University, etc.; and ROBERT J. H. KIMMUTH, Assistant Professor of Physical Education, Yale University. Royal 8vo. Pp. 180 + xiv, with 108 illustrations. 1933. London: Baillière, Tindall & Cox. 23s. net.

AN account of the personal views of the authors upon posture and upon corrective exercises. The latter are not based upon the Ling system, and for this reason alone the book is not likely to be read much in England. Moreover, many of the views on posture would appear to be based on pure mechanics rather than on physiological principles. 'Stretch and strengthen' rather than re-education are emphasized. Many people fail to realize the value of Ling's methods because they have not studied them.

Diseases of the Spinal Cord. By WILLIAMS B. CADWALADER, M.D., Professor of Clinical Neurology, University of Pennsylvania Medical School, etc. Royal 8vo. Pp. 204 + xviii, with 72 illustrations. 1932. London: Baillière, Tindall & Cox. 29s.

THIS book gives an adequate and reasonably compact account of the diseases of the spinal cord. It has no definite surgical orientation and therefore does not demand detailed review here. To those who are called upon to apply surgical methods to cord affections it can be recommended as giving a satisfactory picture of spinal neurology. There is a bibliography of 328 items.

The Digestive Tract. A Radiological Study of its Anatomy, Physiology, and Pathology. By ALFRED E. BARCLAY, O.B.E., M.A., M.D., D.M.R.E., Lecturer in Medical Radiology, University of Cambridge. Large 8vo. Pp. 395 + xxviii, with 275 illustrations. 1933. Cambridge: University Press. 36s. net.

THIS volume is the most complete account of the radiographic appearances of the gastro-intestinal tract that has yet been published in this country. While the book is of special interest to radiologists, for whom the technical side of the matter appears to have been very thoroughly gone into, there are separate sections on the anatomy and physiology of the tract, in which the author elaborates his theory of the fluidity of the abdominal contents, and in which he shows that the position of the stomach varies widely under the influence of various stimuli, even during the course of an examination. Some of our accepted views on the anatomy and physiology of the stomach are bound to be modified by a perusal of this volume, and it may be that the debt which the surgeon owes to radiology will have to be shared in future by his colleagues in the anatomical and physiological schools.

The book is clearly printed, and the numerous radiograms are finely reproduced. The style is fluent and easy, and the author seldom fails to express himself clearly. The book, as a whole, must be regarded as the finest contribution so far made by radiology in this country to our knowledge of the gastro-intestinal tract.

Atlante di Chirurgia dello Stomaco. By G. EGIDI. Large 8vo. Pp. 15, with 30 plates. 1933. Rome: Luigi Pozzi. L. 35.

THIS little atlas of the surgery of the stomach is a credit to the Roman publishing house of Luigi Pozzi. Lying upon the table, it might well be a portfolio of etchings, and indeed the drawings are those of an artist.

A few anatomical plates suffice to make clear the points of entry for injection of anæsthetic fluids, if that method be preferred, and the vascular supply that must be controlled or respected by the operator. One plate displays the sutural methods adapted to gastro-intestinal junctions, and the rest illustrate gastrotomy, gastrotomy, Finney's and Rammstedt's operations, gastro-enterostomy, several types of partial gastrectomy, total gastrectomy, and interventions for gastrojejunal ulcer and gastrocolic fistula. A separate running commentary elucidates the points of the pictures and records the author's predilections as to details.

Roentgenographic Studies of the Urinary System. By WILLIAM E. LOWER, M.D., F.A.C.S., Chief of Department of Urology, Cleveland Clinic, etc.; and BERNARD H. NIENHUIS, M.D., F.A.C.R., Chief of Department of Roentgenology, Cleveland Clinic. Imperial 8vo. Pp. 812 + xviii, with 812 illustrations. 1933. London: Henry Kimpton. 84s. net.

THE rapid advance of urology during the last few years and in particular the development of intravenous pyclography should ensure a welcome for this book. Until recently pyclography could only be carried out with the assistance of surgeons who had the necessary experience in cystoscopy, but the introduction of uroselectan has brought it within the reach of all who can inject the solution into a vein and take skiagrams. The radiology of the urinary tract, however, is not a simple matter; not only are there many essentials in the technique which must be mastered before good and reliable results can be obtained, but also the interpretation of these results is often difficult and calls for considerable experience. Unless the technique is good and the interpretation correct, serious mistakes may be made.

The first part of this book is devoted to an excellent account of the technique of the radiological examination of the various parts of the urinary tract, with a brief description of the pathological conditions and abnormalities which may be found. The second part contains some 750 skiagrams of 443 cases, accompanied by a summary of the history and clinical findings, together with an interpretation of the skiagrams in each case. Most of the skiagrams are admirably clear, and their value is increased by a pen-and-ink sketch illustrating the main points of interest. Some of the intravenous pyclograms, however, are ill defined and might with advantage be replaced by others in the next edition.

There are a few errors—"corpus cavernosa" on page 11, 'hypotonic' for 'hypertonic' on page 55, 'epiploica' on page 61, 'blood' for 'bladder' on page 792, and 'left kidney' for 'right kidney' on page 800b.

This book, written as it is by a urologist and radiologist in collaboration, should prove of great value to those whose experience in urography is not extensive, and will be read with profit by all who are interested in urinary surgery.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

The Thyroid Gland: its Chemistry and Physiology. By CHARLES ROBERT HARRINGTON, M.A., Ph.D., F.R.S., Professor of Pathological Chemistry in the University of London. Large 8vo. Pp. 222 + xiv. Illustrated. London: Oxford University Press. 15s. net.

Intracranial Tumours. By PERCIVAL BAILEY, Professor of Surgery, University of Chicago. Royal 8vo. Pp. 475 + xxiv, with 155 illustrations and coloured plates. 1933. London: Ballière, Tindall & Cox. 35s. net.

Paralysis in Children. By R. G. GORDON, M.D., D.Sc., F.R.C.P. (Ed.), Physician, Bath and Wessex Orthopaedic Hospital, etc.; and M. FORRESTER-BROWN, M.D., M.S. (Lond.), Surgeon, Bath and Wessex Orthopaedic Hospital, etc. Demy 8vo. Pp. 328 + viii, with 116 illustrations. 1933. London: Humpfrey Milford. 15s. net.

Surgery of the Stomach and Duodenum. By J. SHELTON HORSLEY, M.D., F.A.C.S., LL.D., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va. Royal 8vo. Pp. 260, with 136 illustrations. 1933. London: Henry Kimpton. 35s. net.

Actinotherapy Technique. An outline of indications and methods for the use of modern light therapy. With a Foreword by Sir HENRY GAUVAIN, M.D., M.Chir. (Camb.), F.R.C.S. Crown 8vo. Pp. 184. Slough: The Sollux Publishing Co. 6s. net.

St. George's, 1733-1933. By J. BLONFIELD, O.B.E., M.D. Fcap 4to. Pp. 120, with 14 plates. 1933. Published for St. George's Hospital by the Medici Society, London. 5s. net.

- The Enlarged Prostate and Prostatic Obstruction.** By KENNETH M. WALKER, F.R.C.S., M.A., M.B., B.C., Lecturer in Venereal Diseases, St. Bartholomew's Hospital, etc. Second edition. Demy 8vo. Pp. 223 + xiv, with 63 illustrations. 1933. London: Humphrey Milford (Oxford University Press). 14s. net.
- History of Urology.** Prepared under the auspices of the American Urological Association. Editorial Committee: E. G. BALLENGEN, W. A. PHONTZ, H. G. HAMER, B. LEWIS. Two volumes. Royal 8vo. Vol. I. Pp. 386 + xii, with 46 illustrations. Vol. II. Pp. 362 + vii, with 12 illustrations. 1933. London: Baillière, Tindall & Cox. 36s. net.
- Bone Growth in Health and Disease.** By H. A. HARRIS, D.Sc., M.B., B.S. (Lond.), M.R.C.S., M.R.C.P., Professor of Clinical Anatomy, University College and University College Hospital, London, etc. Crown 4to. Pp. 248 + xviii, with 201 illustrations. 1933. London: Humphrey Milford (Oxford University Press). 34s. net.
- Recent Progress in Medicine and Surgery, 1919-1933.** Edited by SIR JOHN COLLIE, C.M.G., M.D., D.L., J.P. With a Foreword by LORD HENDER OF ASHFORD, K.C.V.O., M.D., F.R.C.P. Demy 8vo. Pp. 368 + xii, with 34 illustrations. 1933. London: H. K. Lewis & Co. Ltd. 16s. net.
- The Operative Story of Cleft Palate.** By GEORGE MORRIS DORRANCE, M.D., F.A.C.S. (Philadelphia), assisted by ENAYAT SHIRAZY, D.D.S. Medium 8vo. Pp. 564 + x, with 534 illustrations. 1933. London and Philadelphia: W. B. Saunders Co. 32s. 6d. net.
- The Technic of Local Anæsthesia.** By ARTHUR E. HERTZLER, A.M., M.D., Ph.D., LL.D., F.A.C.S., Professor of Surgery in the University of Kansas, etc. Fifth edition. Large 8vo. Pp. 292, with 148 illustrations. 1933. London: Henry Kimpton. 25s. net.
- The Adrenal Cortex: a Surgical and Pathological Study.** By L. R. BOSTER, O.B.E., M.A., D.M., M.Ch. (Oxon.), F.R.C.S., Surgeon to Charing Cross Hospital; and H. W. C. VINES, M.A., M.D. (Camb.), Pathologist, Charing Cross Hospital Institute of Pathology. Demy 8vo. Pp. 94 + vi, with 4 illustrations. 1933. London: H. K. Lewis & Co. Ltd. 6s. net.
- Rose and Carless' Manual of Surgery.** By CECIL P. G. WAKELBY, D.Sc. (Lond.), F.R.C.S. (Eng.), F.R.S. (Edin.), Surgeon, King's College Hospital, etc.; and JOHN B. HUNTER, J.C., M.Chir. (Cantab.), F.R.C.S. (Eng.), Surgeon, King's College Hospital, etc. Fourteenth edition. Large 8vo. Pp. 1487 + viii, with 721 illustrations and 24 plates (16 coloured). 1933. London: Baillière, Tindall & Cox. 30s. net.
- The Study of Anatomy written for the Medical Student.** By S. E. WHITNALL, M.A., M.D., B.Ch. (Oxon.), M.R.C.S., L.R.C.P., F.R.S. (Canada), Robert Reford Professor of Anatomy, McGill University, Canada. Second edition. Crown 8vo. Pp. 93 + viii. 1933. London: Edward Arnold & Co. 4s. net.
- Pye's Elementary Bandaging and Surgical Dressing.** Revised from H. W. CARSON'S 10th edition of *Pye's Surgical Handicraft* by A. J. CORRINIS, M.B., F.R.C.S., Assistant Director, Surgical Unit, St. Mary's Hospital, etc. Fifteenth edition. Pocket size. Pp. 243 + viii, with 87 illustrations. Bristol: John Wright & Sons Ltd. 3s. 6d. net.
- Diagnostik mit freiem Auge Ekstoskopie und Ektographie mit einem Anhang Tastbefunde.** By Dr. EDUARD WEISZ (Bad Pistyan). Fourth edition. Royal 8vo. Pp. 176 + viii, with 84 illustrations. 1933. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 9.50; bound, RM. 11.
- Röntgendiagnostik der Knochen- und Gelenkkrankheiten.** By Professor Dr. ROBERT KIENBÖCK (Vienna). Large 8vo. Part 1. Differentialdiagnose der geschwulstigen Knochenkrankheiten. Pp. 104, with 26 illustrations. Part 2. Knochenechinokokkose. Pp. 105-192, with 19 illustrations. 1933. Berlin and Vienna: Urban & Schwarzenberg. Part 1, RM. 8.50; Part 2, RM. 7.60.
- The Physician as a Man of Letters, Science, and Action.** By THOMAS KIRKPATRICK MONRO, M.A., M.D., Regius Professor of Medicine in the University of Glasgow. Demy 8vo. Pp. 212 + viii. 1933. Glasgow: Jackson, Wylie & Co. 10s. 6d. net.

THE BRITISH JOURNAL OF SURGERY

VOL. XXI.

JANUARY, 1934.

No. 83.

In Memoriam.

SIR GEORGE MAKINS, G.C.M.G., C.B., LL.D.

MAKINS died on Thursday, Nov. 2, a few hours short of his eightieth birthday.

He was educated at King's School, Gloucester, and entered St. Thomas's Hospital Medical School in 1871. After qualification he studied at Vienna and Halle and worked with Roy at the Brown Institute. He was elected to the staff of St. Thomas's in 1887. He helped Sir William MacCormac in the organization of the International Medical Congress in 1881 and was joint treasurer with Sir Dyce Duckworth of that held in 1913.

He was Consulting Surgeon in the Boer War, and embodied his experiences in a book which was translated into several languages. In the Great War he was again a Consulting Surgeon. In 1917 he came home to be President of the Royal College of Surgeons—a position that he filled with great distinction. In the same year he went to India to report on the British Station Hospitals.

He was an active member of the British Red Cross and of the Nightingale Fund, Treasurer of the Imperial Cancer Research Fund, Chairman of the Distribution Committee of the Hospital Sunday Fund, and Chairman of the Executive Committee of the Athenæum.

These bare details of his life show the diversity of his interests and the widespread nature of his influence. Makins by his work earned for himself a fine position in surgery; but he had a greater success than this in that he won the regard and esteem of those around him no matter whether they were old or young. His appearance was striking and dignified; his smile was wonderfully attractive. He had a great store of wisdom, often expressed in unusual terms which were very apposite. This wisdom concerned not only surgery but the everyday things of life. If you went to consult him in an anxious and perturbed state of mind, you soon found things resolving themselves to their just proportions and your course of action seemed obvious, if not agreeable. To be with him gave a sense of peace and security. He was often late, never in a hurry, and yet accomplished with ease all he had set out to do.

As a surgeon he was sure, imperturbable, and constructive; friends, nurses, and doctors owe him much. The confidence in his judgement can

be told by the number of committees on which he served and where his counsel was sought and valued.

Quite unselfish, quiet in argument, which he disliked, he could be very firm but was always fair. In matters affecting himself he was much less tenacious, and on occasion gave way to another, though from the amusing and tolerant way in which he described the retirement one felt that he had not altogether had the worst of the encounter.

In his youth he was rather apart and showed but little aptitude for games. At an early age he took to fishing and gardening. Later on he became a climber, sharing the dangers and delights with Lady Makins. He was a lover of music and played the cello, but flowers were his chief hobby, and it was delightful to wander round and hear him talk about them. He was fond of company and a charming host, and many will look back with genuine pleasure to the hospitality of Lady Makins and himself.

Conversation was no trouble to him. He had met many folk, and few great men in the medical world were unknown to him; many had stayed at his house. He had retained kindly memories of these people which he was wont to recall in his quiet and distinctive voice. He was a man around whom stories gather, and the tradition of Makins will long remain at his hospital.

To the very end he remained the same, a humorous, placid, kind, and fearless man. A fine life and bravely ended.



SIR GEORGE MAKINS, G.C.M.G., C.B., LL.D.



PRESENTATION TO THE EDITORIAL SECRETARY.

At a Meeting of the Editorial Committee held on Oct. 12, 1933, reference was made to the completion of the 20th volume of the JOURNAL, and the occasion was marked by a presentation to Mr. Hey Groves, who has filled the office of Editorial Secretary since the founding of the JOURNAL.



Lord Moynihan, in making the presentation, said :—

“MR. HEY GROVES :

“This presentation is made to you on behalf of a number of subscribers to the BRITISH JOURNAL OF SURGERY in recognition of your invaluable services during twenty years as Editorial Secretary. We offer you this salver and cheque as a tribute to your efficiency and devotion. You were one of those in whose mind was conceived the idea of founding in this country a journal worthy of the contributions to the Art and Science of Surgery which England and the British Empire were making. Difficulties were many, and opposition and indifference were found in high places. But we were not dismayed, and

faith in our venture was fired by this experience. We neither faltered nor wavered, but held steadily to our purpose. In earliest days and throughout twenty years, with the exception of one year when you served in the Army oversea, our activities centred round you. All our plans, all our ambitions, all our efforts, found in you a most willing, most competent, and tireless worker. Your industry, your wise judgement, your enthusiasm and unwearying tact, have been of value beyond reckoning in maintaining year after year our corporate efforts. The doubts and hazards of early years are now displaced by an assured confidence in our work, and hope for the future of the JOURNAL is now secure.

‘Incertainties now crown themselves assured’.

“If our JOURNAL is of value to the world of surgeons, to no man can greater credit be given than to you, our faithful, loyal, most diligent and trusted Secretary. To-day and by our simple gift we seek with our affection to assure you of our cordial recognition of your invaluable help, and to say of you and the JOURNAL—

‘So long as man can breathe or eyes can see
So long lives this, and this gives life to thee.’”

SOME EARLY SURGICAL CASES.

BY SIR D'ARCY POWER, K.B.E., LONDON.

II. THE EDWIN SMITH PAPYRUS.*(Concluded.)*

PASSING from head injuries, the surgeon next describes broken noses, first where the cartilage alone is injured, and secondly when the nasal bones are fractured. Of traumatic deviation of the septum, he says:—

Instructions concerning a break in the column of the nose. If thou examinest a man having a break in the column of his nose, his nose being disfigured and a depression being in it while the swelling that is on it protrudes and he has discharged blood from both his nostrils. Thou shouldest say concerning him; "One having a break in the column of his nose. An ailment which I will treat."

Thou shouldest cleanse it for him with two plugs of linen. Thou shouldest place two plugs of linen saturated with grease in the inside of his two nostrils. Thou shouldest put him at his mooring stakes until the swelling is reduced. Thou shouldest apply for him stiff rolls of linen by which his nose is held fast. Thou shouldest treat him afterwards with grease, honey and lint every day until he recovers.

CARCINOMA OF THE KIDNEY.

By GRAHAM SIMPSON,

SENIOR SURGEON, ROYAL HOSPITAL, SHEFFIELD.

SINCE the classical paper by Grawitz in 1883, much has been written on the diagnosis of renal hypernephroma and on the vexed question of its pathology: this is probably the reason that the interest shown in the other neoplasms has suffered from a temporary eclipse.

A study of the literature proves that, though carcinoma of the kidney is recognized as a sufficiently well defined form of renal growth, its description is limited usually to specimens obtained by operation or at autopsy, and little or no attempt is made to distinguish it clinically.

In a recent text-book on surgical pathology the author divides renal growths into two groups: *the hypernephroma or Grawitz type* and *the embryoma or Wilms type*, and he states that "It will be found that the vast majority of tumours encountered in actual practice can be placed in one or other of these groups". In my opinion such a statement is misleading. Whilst it is unlikely that many cases of carcinoma of the kidney will come within the ken of any general surgeon, recent experience has convinced me that these tumours are far from uncommon: moreover, they may present certain quite characteristic symptoms, the recognition of which is of great importance: otherwise, some patients will undergo a dangerous and unnecessary operation.

Before discussing the symptoms incidental to this form of growth and the complications peculiar to it, I will give the history of a case in which I was able to make such a diagnosis and to predict, with some accuracy, the future of the patient.

CASE REPORT.

Mrs. H., aged 60. In October, 1930, I was asked to see her for subacute intestinal obstruction: she had been poorly since June and had complained of constant severe pain in the back.

She was admitted to a nursing home, where a turpentine enema at once relieved the obstruction, which was obviously due to faecal impaction; a barium enema was then given and the colon shown to be normal.

On going more closely into her history, she recalled that, some two years before this, she had suffered from a sharp hæmaturia which had continued for several days. Her medical man was just about to ask me to see her when the bleeding ceased, the consultation was cancelled, and the patient went away for a holiday. She had never noticed blood in her urine since that one occasion.

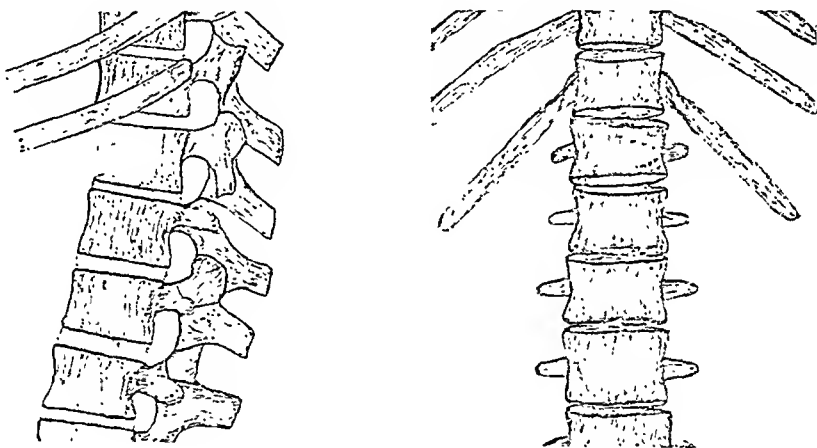
The distension of the abdomen had now subsided, and I again examined her, but I was unable to feel either kidney and there was no tenderness in either loin. The urine was clear and contained neither blood nor pus, but there was a well-marked cloud of albumin.

I now ventured to suggest to her doctor that this woman was suffering from a carcinoma of the kidney, though which organ was affected I could not say; that a

cystoscopy would reveal that one kidney was out of action; that an X-ray examination would demonstrate disease of one or more of the lumbar vertebrae; and that she would become paraplegic and die in a very short time.

A chromocystoscopy showed a normal bladder and ureteric orifices; from the right ureter blue appeared in three and a half minutes, but none came from the left in fifteen minutes. The X-rays showed caries of the body of the first lumbar vertebra with partial collapse (*Figs. 265, 266*). The pain in the patient's back continued to be so terrible that it was with difficulty controlled by morphia; she rapidly went downhill, soon became unable to move her legs, and died six weeks after I first saw her.

I obtained consent for an autopsy, and the illustration (*Fig. 267*) represents the left kidney and part of the dorsal and lumbar spine. The kidney, though almost entirely replaced by growth, is hardly larger than normal; it measures $4\frac{1}{2}$ in. (11.4 cm.) in length and $2\frac{1}{2}$ in. (6.3 cm.) in breadth; very small portions of the kidney can still be made out at the lower pole and on the outer side.



FIGS. 265, 266.—Showing caries of body of first lumbar vertebra and partial collapse.

Around the pedicle is a mass of firm fibro-fatty tissue in which the renal pelvis is buried and cannot be seen; in this mass is included the adrenal, which is also infiltrated with growth; this fibro-fatty mass extended to the side of the vertebral body, but was not firmly adherent to it.

The body of the 1st lumbar vertebra is largely replaced by growth; at this level the spinal column was quite soft and could be freely bent; as a result of this softening the intervertebral discs above and below had been pressed together. There were also some deposits in the body of the 2nd lumbar vertebra.

Sections were cut of the left kidney (*Fig. 268*) and of the body of the 1st lumbar vertebra (*Fig. 269*), and they show that the growth is a typical infiltrating carcinoma. There is no attempt at the formation of tubules, but in various places the arrangement of the cells and their staining reactions strikingly recall the structure of a carcinoma of the bladder; possibly this tumour started in the transitional epithelium of the pelvis or calices.

REMARKS ON THE DIAGNOSIS AND PATHOLOGY.

During the last five years three similar cases have come under my care, and in each of them the clinical picture was so striking as to be quite characteristic:—

some of the modern pathologists. There can be no doubt that, from a study of their minute structure, it is impossible to draw any absolute line between them; in other words, the Grawitz tumour does not 'breed true'. Nevertheless, in spite of the difficulty in distinguishing them microscopically, I consider that an attempt should be made to keep the old nomenclature.

The Grawitz tumour can be recognized clinically and by naked-eye examination; it has a characteristic colour, it is at some stage of its growth encapsulated, and, if diagnosed early, gives an occasional brilliant surgical success. The infiltrating carcinoma also has its characteristic mode of growth,



Fig. 270.—Showing invasion of right half of 1st lumbar vertebra.

shows no sign of encapsulation at any period of its existence, and is much more fatal.

Great emphasis has of late been laid on the importance of *haematuria*; the vital necessity of considering this as a symptom and not as a disease is fully supported by these cases. In every one of these patients blood in the urine was the first sign of there being anything amiss; usually it had appeared a long time before I saw the patient and had never recurred.

The *pain* is very intense, and is generally referred to the lumbar spine; in one case it was felt also in the course of the external cutaneous nerve of the thigh. In only one of my cases was it felt in the loin as well as in the

back. Since it is not felt along the nerves of the lumbar plexus it is unlikely that it is caused by compression of these nerves in the spinal foramina. It may be compared to the pain which accompanies compression fractures of the vertebræ or to that complained of by patients with carcinoma of the breast who are the subject of secondary deposits in the spine. It is a curious fact that the erosion of the vertebral bodies with subsequent collapse due to tuberculosis is not associated with much local pain, though a typical girdle pain may be present.

The most striking finding in this group of cases was the constant presence of *collapse of one or more of the vertebral bodies*; in each case this was shown by the X-rays; in the 1st lumbar in two cases and in the 2nd and 3rd in the others. The strict localization to the three upper lumbar vertebræ suggests a local spread, presumably through the lymphatics. This view is supported by a case published by Professor John Fraser, of Edinburgh, to whose courtesy I am indebted for permission to reproduce *Fig. 270*. In this case, also one of carcinoma of the kidney, only that half of the vertebral body is affected which is on the side of the growth.

Another noticeable feature is the early onset of a profound *cachexia*; this has been mentioned by Israel as characteristic of renal carcinoma.

TREATMENT.

At this stage of the disease attempts to relieve the terrible pain in the back seem to be the only indication; one patient required 6 gr. of morphia in the twenty-four hours to make life at all tolerable. What is to be done in the future for these unhappy individuals?

In a recent speech Sir Buckstone Brown said that Nature's method was "a word and a blow", and it would be hard to find a more apt example than the mode of onset of this disease: the word of warning, the symptomless hæmaturia, followed months or years later by the pain in the back which makes the diagnosis simple and treatment hopeless.

If, as it seems to me, the one hope lies in earlier diagnosis, our students should be taught to look on hæmaturia as a surgical emergency. If this were done and such patients sent at once to a nursing home or a hospital for immediate cystoscopy, we might occasionally come across a carcinoma of the kidney in time for a successful operation. At the present time nephrectomy for such tumours does little more than enrich the local pathological museum.

Unfortunately the hæmaturia precedes the pain and it may be difficult to persuade the patient to submit to the inconvenience of a cystoscopy. In some cases, owing to delay in getting a bed, the bleeding has stopped before the patient is admitted, and either a cystoscopy is refused or it is impossible to tell from which side the bleeding came.

Great hopes have been raised by the advent of pyelography and its more recent developments, but I doubt whether the original method of 'infusion pyelography' will be of much help in early cases of this disease. Even in advanced cases there is little deformity of the kidney and its pelvis, and it is common knowledge that the pelvis and calices of normal kidneys show great variation in outline. Not only so, but the taking of bilateral pyelograms

and the comparison of the suspected kidney with that of the opposite side may mislead. for, in my experience. the arrangement of the pelvis and calices is not always symmetrical. If to this we add the possible presence of filling defects due to blood-clot and the effects of over- and under-distension, I feel I am justified in my pessimism.

There is, however, a brighter side to the picture ; the use of uroselectan, 'excretion urography', though it may fail to demonstrate the form of the pelvis and calices, almost certainly is an indicator of the function of the kidney. In a recent paper on this subject. Mr. Henry Wade, of Edinburgh, asserts that, whilst the infusion method may fail to help in the diagnosis of this form of growth. the excretion method may show an entire absence of shadow. I have not yet had the opportunity of confirming his observations, but they seem to me to be the most hopeful yet made on the diagnosis of this disease.

I feel that this is a real advance. for there are many patients, especially men, who would consent to an intravenous injection but would object to a cystoscopy which might be prolonged and might have to be repeated. Then again the injection can be made by a resident and does not demand the skill necessary for cystoscopy.

Even if the patient has been cystoscoped at once and the bleeding traced to one kidney. our difficulties are not at an end, for a surgical problem of some delicacy arises when the suspected kidney is exposed on the loin and is found to look and to feel perfectly normal.

There can be no doubt that. if such an organ is stripped of its capsule, or if it is fixed in the loin, or if it is simply returned to its original site, a certain number of these cases cease to bleed and are reported to be in good health many years later. I have not adopted this line of treatment, for I felt that, sooner or later, I should in this way miss a case of early tumour.

Another method is to split the kidney longitudinally and examine the cut surfaces and the pelvis and calices ; they can be inspected quite efficiently if the renal pedicle is compressed. After exploring a good many kidneys in this way I gave it up because I consider there are three grave disadvantages.

1. The first and greatest is that, in spite of careful suture of the two halves, I have had several instances of very severe hæmorrhage a few days later, and this has occurred notwithstanding the fact that, at the subsequent nephrectomy, the sutures were still holding.

2. The second is that such a kidney after a secondary removal may show numerous large infarcts—a proof that the blood-supply has been seriously interfered with. I feel doubtful whether such damaged organs are of much use afterwards.

3. The last is that one might replace a kidney which was the seat of an early growth ; even the hemisection shows only two surfaces, and a small tumour to one or other side of the plane of section might conceivably be missed by the most careful of surgeons.

Still another and more radical procedure is to do a nephrectomy, and to this course I have been driven by the rule that I have formulated for myself that "more can sometimes be learnt of the pathology of a kidney by the examination of its separated urine than by its exposure on the loin".

Perhaps the best example of this is the early tuberculous kidney, which looks normal when exposed at operation but the urine from which has been found to contain pus and tubercle bacilli.

My practice for some years has been to remove the kidney, have it hardened, and to open it and have it examined some weeks later. In this way I have met with some interesting findings—a nævus of a papilla and very early tubercle of one papilla. My colleague, Mr. J. C. Anderson, has recently published in this JOURNAL an example of a tiny papilloma of a papilla which gave rise to sharp bleeding. The following is a still more striking case:—

In October, 1930, a man aged 37 was admitted under my care for hæmaturia of two weeks' duration: his only other complaint was of pain in the left iliac fossa which was felt on passing water.

He looked quite healthy and nothing abnormal was found on examination except the bleeding, which was traced to the right kidney. This was removed; it looked normal, but the pelvis and ureter were distended with blood.

On cutting into the kidney the uppermost calix was found to be full of blood and the two highest papillæ had a peculiar velvety appearance. Sections were cut of these papillæ and they show a curious change in the tissue immediately under their tips; the accompanying illustration (Fig. 271) shows that they closely resemble a section of hypernephroma. The change was limited to the two upper papillæ and was not present in the papilla just below.



FIG. 271.—Section of papilla.

I hesitate to call this a case of very early tumour, for the changes are slight and confined to so small a part of the organ; moreover, Dr. Harold E. Harding very kindly studied the sections for me and his report (which is given at the end of this paper) is definitely against the view that these changes are neoplastic.

Nevertheless, in spite of the dubious nature of the lesion, the bleeding was severe, and I feel that I was justified in my choice of treatment; I am confident that many surgeons, on looking at this section, would feel little doubt that it was from a hypernephroma. It is hardly necessary to add that the patient is alive and quite well.

Leaving aside for the moment the question whether nephrectomy for symptomless hæmaturia is a sound form of treatment, I must admit that there are some tumours of the kidney which are perfectly hopeless in the present state of our knowledge. The following case is an example:—

Boy, aged 17, admitted to the Sheffield Royal Hospital for hæmaturia of only three days' duration: he had had no previous symptoms and looked to be in perfect

health. The bleeding was traced to the left kidney, and this was removed four days later; the pelvis and the lower pole were noticed to be unusually adherent; otherwise the organ looked quite normal. (Fig. 272.)

The kidney was hardened, and when it was cut across a cyst was found in the lower pole containing blood and clot; projecting into its cavity was a polypus, the size of a cherry-stone; a section of this showed spindle-celled sarcoma.

The growth recurred *in situ* within six months, and on readmission of the patient was found to be inoperable; it spread with frightful rapidity until it filled half the abdominal cavity, caused the most intense suffering, and destroyed the patient in a few weeks.

This rapid recurrence was the more surprising as the kidney had been removed intact and only cut open some weeks later, so that the question of the soiling of the wound with tumour cells can hardly be entertained.

Such a lamentable result after removal of a tumour only four days after the first symptom makes one wonder whether we are justified in telling the public that the cure of cancer is only a question of early diagnosis.

But there is another side to the question of nephrectomy for symptomless hæmaturia: I have to admit that, on several occasions, I have removed such kidneys and that nothing abnormal has been found after the most careful examination to account for the hæmaturia.

On the whole, my experience in the search for early kidney tumours has been disappointing. I have removed some kidneys which might presumably have stopped bleeding and not given any future trouble. I have removed a kidney seven days after the first bleeding and the patient was dead of a sarcoma in six months.

The tumours I have found have been mostly

innocent and might perhaps have been cured by less radical methods than nephrectomy, and the one case in which I thought I had discovered an early hypernephroma is not considered by the pathologist to be neoplastic at all.

Nevertheless, I have thought it worth while to record my experience, if only to direct the attention of other surgeons to this very difficult and unsatisfactory problem.

HISTOLOGICAL REPORT.

BY HAROLD E. HARDING.

The cells of the collecting tubules are large and columnar. The rounded nuclei are rather darkly staining; most of them have a single nucleolus and a dense reticulum; they are nearer to the basement membrane than to the margin of the cell.



FIG. 272. -Sarcoma of kidney.

The cytoplasm is extremely thin, so that a well-marked cell membrane appears to enclose a space very imperfectly filled with a little finely granular cytoplasm, and in which the nucleus has no visible means of support. The appearance of these cells is not unlike that of the so-called 'hypernephroma'. Obviously a large proportion of the cell content has been dissolved out in preparation of the section.

These appearances are most marked at the inner surface of the renal pyramid, but can be traced inwards for at least a quarter of an inch, the cells becoming rather less columnar and more cubical.

In most cases the tubules have only a single layer of epithelium, but cutting on the slant at places gives a layered appearance. In the largest tubules, however, the epithelium becomes two or three cells thick and there is a notable tendency for papillary projection into the lumen of the tubule. In every case the cells of the collecting tubules are confined within their basal membrane.

I do not consider the condition to be neoplastic.

RIGHT PARADUODENAL HERNIA.

BY A. C. HALLIWELL.

SURGEON, JERSEY GENERAL HOSPITAL.

THERE are two types of hernia into peritoneal fossæ in the neighbourhood of the duodenojejunal flexure, the right and the left paraduodenal. Of these, the former is by far the rarer, only 35 cases having been recorded, whereas there are 105 cases of the left paraduodenal hernia in the literature. Of the 9 peritoneal fossæ in this region described by Lord Moynihan,¹ only 2 or possibly 3 are of surgical importance. These are:—

1. *Left Paraduodenal Fossa of Landzert*.—This is situated on the left side of the duodenojejunal flexure, and the orifice looks towards the right. It is a pouch of peritoneum pushed beneath the inferior mesenteric vein, which consequently lies in its right free margin, and a hernia in this fossa is known as a 'left paraduodenal' hernia.

2. *Right Mesenterico-parietal Fossa of Waldeyer*.—This is situated below the duodenum and is bounded in front by the mesentery of the jejunum. Its orifice looks towards the left, and the superior mesenteric artery lies in its left free margin. Moynihan, writing in 1906, stated that he had only met with a distinct fossa in adults on three occasions, but in seventeen embryos of less than five or six months he noticed six fossæ. It is believed that the majority of cases of right paraduodenal hernia are in connection with this fossa.

3. *Inferior Duodenal Fossa of Treitz*.—Klob, who first described right paraduodenal hernia in 1861, believed that the hernia entered the inferior duodenal fossa, which lies at the left side of the termination of the duodenum, and progressed towards the right by peeling back the superior peritoneal margin until the superior mesenteric artery is reached. This would produce a typical right paraduodenal hernia. Nagel² considers that some of these herniæ probably do arise in this way, although the majority are in connection with the mesenterico-parietal fossa. The inferior duodenal fossa is common, being found in 60 to 70 per cent of cases.

Moynihan states that the following conditions are invariably present in right duodenal hernia: (1) The sac occupies, at any rate at first, the right half of the abdomen, lying behind the ascending and transverse mesocolon; (2) The orifice is situated behind and to the left of the sac; (3) In the anterior margin of the sac is either the superior mesenteric artery or its continuation, the ileocolic artery. In the case described below these three conditions were present.

The literature on this variety of duodenal hernia was reviewed by Nagel² and again by F. R. Brown,³ who summarized the 32 cases recorded to that date. In 1929 Taylor⁴ added 2 more and described the first case to be

diagnosed before operation or necropsy. A further case was reported by Dowdle⁵ in 1932. Of these 35 cases, 18 were operated upon, the remaining 17 being found at necropsy. There have been only 7 recoveries from the operation.

CASE REPORT.

A. J., a man aged 38, was admitted to the Jersey Dispensary and Infirmary in May, 1932, with twelve months' history of pain across the lower abdomen. The pain was colicky in nature and was brought on by food, appearing usually about half an hour after meals. At the beginning of his illness there were days of complete freedom from pain, even as long as two weeks, but during the two or three months previous to admission it occurred after every meal and lasted

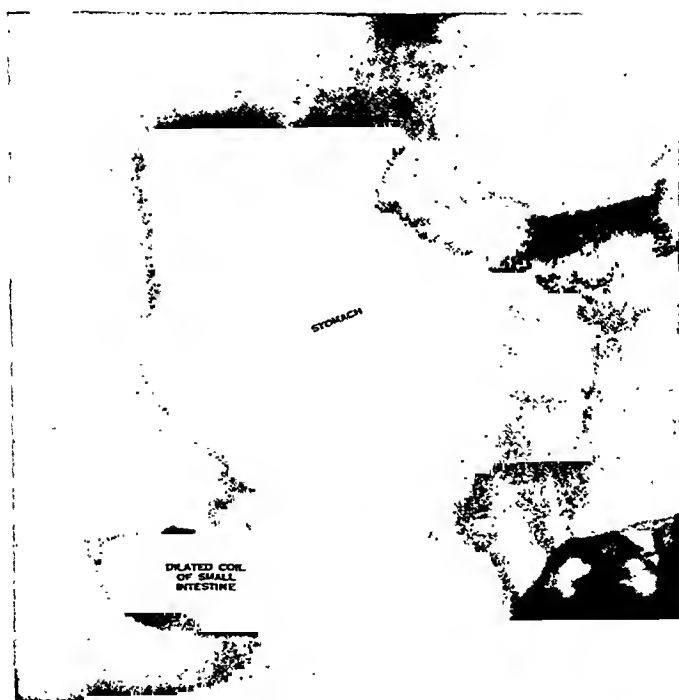


FIG. 273.—X-ray appearance immediately after meal.

till he vomited, after which the pain was relieved. His appetite was good, but he was afraid to eat.

ON EXAMINATION.—The patient appeared well nourished. There was slight abdominal distension, and in the upper left quadrant visible peristalsis was noted. X-ray examination showed dilated coils of small intestine bunched together, chiefly on the right side of the abdomen (*Figs. 273-277*).

DIAGNOSIS.—A diagnosis was made of chronic small intestine obstruction, probably retroperitoneal hernia. After the operation, when the films were compared with those published by Taylor¹ in the *BRITISH JOURNAL OF SURGERY*, it was felt that the diagnosis should have been made more accurately.

OPERATION.—The abdomen was opened by a right paramedian incision and revealed the condition shown in the drawing (*Fig. 278*). There appeared to be three large loops or coils of bowel and no small intestine visible anywhere. These coils overlapped the ascending and descending colon. The membrane covering the loops was moderately thick, and although the individual coils of small intestine could be seen through, they were not as clear as appears in *Fig. 278*. We were obviously dealing with a retroperitoneal hernia, and the origin was found in the neighbourhood of the duodenojejunal flexure. The mouth of the sac looked towards the left, and over it the superior mesenteric artery could be felt. An attempt was

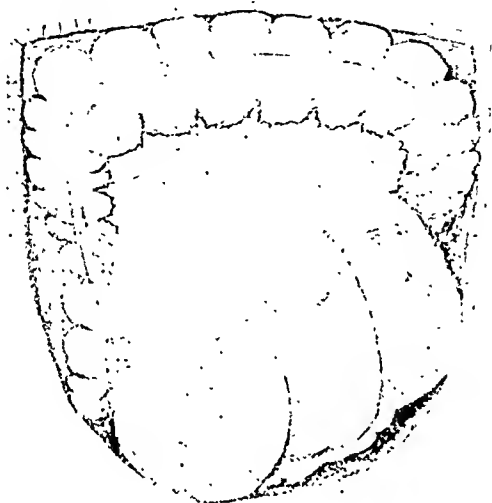


FIG. 278.—Condition found at operation.

made to withdraw the bowel, but this was found to be impossible owing to adhesion of the entering and returning loops at the mouth of the sac; consequently an opening was made into the front of the sac to investigate the condition inside. There was only one layer of peritoneum, and the serous coat of the bowel was completely adherent to the wall of the sac. It could be peeled off without difficulty, but left an intestine devoid of its serous coat; consequently the hole made in the sac was closed. The neck of the sac was examined again and divided on its inferior aspect, freeing the entering and returning loops as far as possible, after which the abdomen was closed.

SUBSEQUENT PROGRESS.—There was a good deal of post-operative vomiting, and for a few days it appeared as if the chronic obstruction had been converted into an acute one. Finally, however, the patient made a good recovery, and nine months later was reported as at work and free from symptoms.

COMMENTARY.

Although the patient cannot be considered cured, he has been relieved of his symptoms by division of the neck of the sac, and indeed it is difficult to see what else was possible. Brown was able to turn the sac inside out, ligature it at the neck, and remove it. In this case there was no sac and it was not a true herniation. The obvious suggestion is that the intestine had burst through the wall of a small sac and had insinuated itself behind the posterior parietal peritoneum. But there are many objections to this theory,

and these have been clearly stated by Edmund Andrews,⁶ who maintains that the term 'duodenal hernia' is a misnomer and that the condition is a "congenital anomaly due to imprisonment of the small intestine beneath the mesentery of the developing colon". His arguments are convincing and well bear repetition. It is difficult to imagine a force which will continue to act until all the small intestine has been segregated into a sac, leaving the abdominal cavity empty. There are many similar folds and fossæ in the peritoneal cavity, some of which are larger than the duodenal fossæ, and are practically never the site of a hernia. The herniated viscus is always small intestine, and practically the whole of the small intestine: the presence of omentum has never been reported. Finally a case of total hernia has been reported by Vogt in a new-born infant (left duodenal).

The case reported above is consistent with an embryological explanation.

I am indebted to Dr. Stamberg, Honorary Radiologist to the Jersey Dispensary and Infirmary, for the series of X-ray photographs.

REFERENCES.

- ¹ MOYNIHAN, *Retroperitoneal Hernia*, 1906, 2nd ed.
- ² NAGEL, *Collected Papers of the Mayo Clinic*, 1923, xv, 135.
- ³ BROWN, *Brit. Jour. Surg.*, 1925-26, xiii, 367.
- ⁴ TAYLOR, *Ibid.*, 1929-30, xvii, 639.
- ⁵ DOWDLE, *Surg. Gynecol. and Obst.*, 1932, liv, 246.
- ⁶ ANDREWS, *Ibid.*, 1923, xxxvii, 740.

TWO CASES OF OSSIFICATION IN THE INTERNAL SEMILUNAR CARTILAGE.*

By H. JACKSON BURROWS.

CHIEF ASSISTANT, ORTHOPÆDIC DEPARTMENT, ST. BARTHOLOMEW'S HOSPITAL.

THE following two cases seem to be of interest as examples of a rarity and as bearing on the problem of the metaplastic formation of bone.

CASE REPORTS.

Case 1.—F. R., Eastern produce merchant, aged 25. Two and a half years previously the patient was running at Rugby football, and as his weight came on the right foot this slipped inwards. He felt a sudden pain internal to the right patella, and was unable to continue the game. The knee became swollen about six hours later, and his activities were limited for three or four days, after which



FIG. 279.—*Case 1.* Skiagraphs showing opaque body in joint.

the joint felt perfectly recovered. Subsequently, however, the symptoms returned on six or seven occasions. At no time did locking occur.

The only physical sign at the time of examination was slight tenderness in the joint line internal to the patella.

X-ray photographs (*Fig. 279*) showed an opaque body lying in the postero-internal part of the joint.

* From the Orthopædic Department, St. Bartholomew's Hospital, and the Cytological Laboratory, Royal College of Surgeons of England.

A diagnosis of loose body (probably due to osteochondritis dissecans) was made.

OPERATION.—The joint was explored by the postero-internal route. It was found that the posterior horn of the internal semilunar cartilage was absent, the latter ending posteriorly in a club-shaped extremity which was 1 cm. broad and $\frac{1}{2}$ cm. thick, continuous in front with the remainder of the cartilage, but otherwise quite free. It was faceted below by the articular surface of the head of the tibia, was bony hard, and had a pale pink tint which contrasted with the rest of the meniscus. An antero-internal incision was then made and the whole cartilage removed in one piece: the anterior portion showed no abnormality.

Subsequent examination of the specimen (*Fig. 280*) confirmed the above description. The histology is detailed later.

Case 2.—B. W., clerk, aged 19. Five years previously the patient had been kicked behind the right knee at football. He continued the game, but afterwards noticed stiffness, and swelling occurred which lasted about a month. Subsequently at football or at other times, when he turned inwards on the right leg, there would be a sudden pain in the postero-external part of the joint which would let him down. This would be followed by aching pain lasting for a period of one to three days. There was never any locking.

No physical signs were present at the time of examination except a click of uncertain origin in the postero-external part of each knee on extension after full flexion.



FIG. 280.—Case 1.
Specimen viewed from above.



FIG. 281.—Case 2. Skiagraphs showing opaque body in joint.

Skiagraphs (*Fig. 281*) showed an opaque body lying in the postero-internal part of the joint, and a like condition at re-examination four weeks later after splinting prior to operation.

A diagnosis of loose body (probably due to osteochondritis dissecans) was made.

OPERATION.—The joint was opened by a postero-internal incision. The condition of affairs was similar to that found in *Case 1*, except that the abrupt termination of the cartilage consisted of two parts: (1) A hard, club-like, smooth nodule similar to that found in *Case 1* (except in being faceted above instead of below); and



FIG. 282.—*Case 2*. Specimen showing (a) superior, and (b) inferior surface.

(2) A ragged tag of fibrocartilage which lay below and slightly internal to the nodule and was separated from it by an irregular split (*Fig. 282*). As much of the cartilage was removed as was possible without making a further incision. About the anterior third remained.

The findings at operation were confirmed by subsequent examination of the specimen.

HISTOLOGICAL EXAMINATION OF THE SPECIMENS.

Histological examination in *Case 1* showed that the hard nodule contained bone (*Fig. 283*). The fibrocartilage immediately surrounding this is

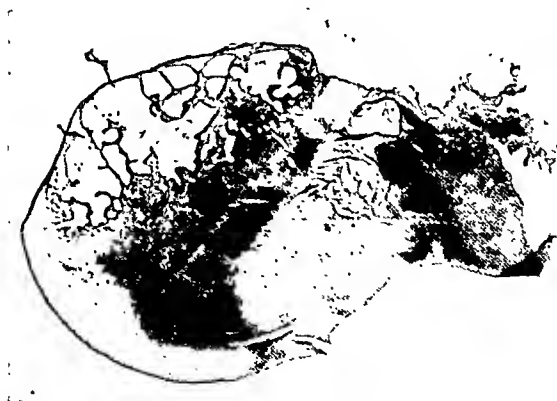
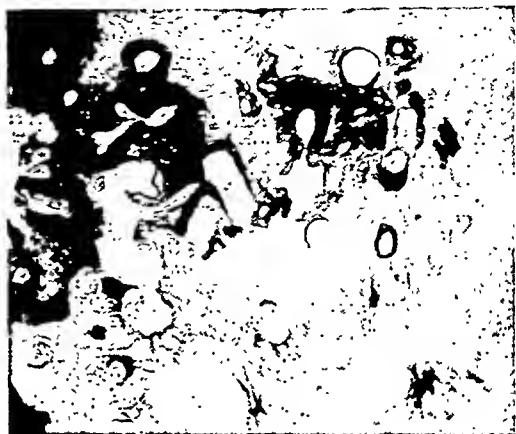


FIG. 283.—*Case 1*. Section of specimen. Note spongy bone above and to the left. (Zenker's fixative; Ehrlich's haematoxylin and eosin.) ($\times 5$.)

in places clearly demarcated from the trabeculae; in other parts there is some tendency to a blending or transition. It shows patches of calcification, and

its cells are increased in number and hypertrophied. Many of them are surrounded by chondrin-balls. While this tissue retains the essential characters of fibrocartilage, its appearance approximates to that of hyaline cartilage. The portion immediately contiguous to the main nodule of bone contains spaces occupied by loose connective tissue and blood-vessels (*Fig. 284*). Some

FIG. 284.—*Case 1.* Section of specimen to show the area adjacent to the main nodule of bone. Note the tendency for the early bone to form round the fibrovascular spaces which have appeared in the hyperplastic cartilage. (Zenker's fixative; Mallory's triple stain.) (*Fig. 35*.)



of these are surrounded or partly surrounded by a thin lamella of bone. Osteoclasts are inconspicuous. The sections suggest that the process of bone formation is as follows:—

1. Hyperplasia of the fibrocartilage, with deposition of calcium; the cells multiply and enlarge and the proportion of matrix to fibres increases.
2. Replacement of areas of cartilage by loose vascular connective tissue.
3. Formation of true bone in the walls of the cavities so formed.



FIG. 285.—*Case 2.* Section of specimen. (Zenker's fixative; Mallory's triple stain.) (*Fig. 35*.)

The appearances in *Case 2* (*Fig. 285*) resemble those in *Case 1* in that the fibrocartilage contiguous to the bone has undergone hyperplasia. They differ in the following particulars. Vascular connective-tissue spaces are relatively absent from the zone of hyperplastic cartilage. In parts the cells

are particularly numerous and may tend to become arranged in rows at right angles to the zone of ossification (*Fig. 286*). The nodule of bone contains

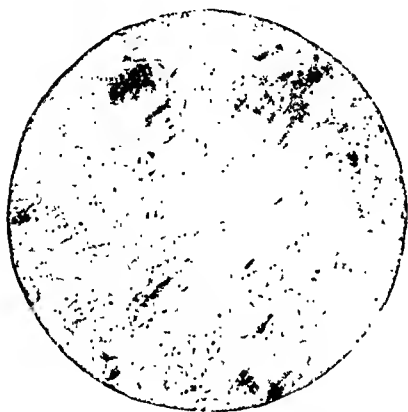


FIG. 286.—*Case 2.* Section of specimen to show an area where the numerous cells of the hyperplastic cartilage have become arranged in rows at right angles to the zone of ossification. (Zenker's fixative; Mallory's triple stain.) ($\times 20$.)

several small islands of hyaline cartilage (*Fig. 287*), and there is a thin shell of this overlying a part of the surface.



FIG. 287.—*Case 2.* Section of specimen to show islands of hyaline cartilage. Note the fibrous appearance typical of young bone. (Zenker's fixative; safranin and picro-indigo-carmin.) ($\times 60$.)

COMMENTS.

Clearly considerable caution must be exercised in drawing conclusions from only two cases.

Symptoms, Diagnosis, and Treatment.—Both patients are young men who had received an injury at football a few years previously. Each had been troubled by a return of symptoms from time to time, with apparent

complete recovery in the intervals. Locking had been absent. X-rays showed an opaque body of about the same size in about the same position in each instance.

These points might assist a correct pre-operative diagnosis on another occasion, and might be thought to justify an initial antero-internal incision, through which all that is required could be done. It would be much safer, however, as such a diagnosis must always be a tentative one, to proceed in the manner customary for a loose body in the situation named. Whether an antero-internal incision should subsequently be made and the anterior part of the cartilage removed is a matter of opinion. As yet, it is too soon to know whether the fact that this step was omitted in *Case 2* will give rise to trouble. Personally I should prefer to do the more complete operation.

Pathology.—The possibility that a preceding congenital abnormality was present cannot be denied.

That the condition is in part, if not wholly, traumatic is suggested by the history and supported by the fact that the second specimen showed all the appearances typical of an old split; both showed histological evidence of osteogenic activity, which would be improbable if the bone had dated from early life.

If the condition is wholly traumatic, the bony nodule in each case must be metaplastic. From the description of the histological appearances in *Case 1* it is clear that in this instance there is a close resemblance between the process of metaplastic bone formation in fibrocartilage and the normal process of ossification in hyaline cartilage. *Case 2* also is consistent with this, though giving a less complete picture. The islands of hyaline cartilage in this case may be compared with those sometimes found in heterotopic membrane bone. They differ from the residual cartilage found in areas of recently formed enchondral bone and cannot necessarily be regarded as a stage in the change from fibrocartilage to bone.

The present example of metaplastic bone formation is particularly interesting because it is enchondral. Most examples which have received study have represented ossification of the membrane type.

From histological considerations it would seem that the crucial event is the hyperplasia of the fibrocartilage. In embryonic skeletal rudiments the onset of ossification is preceded by the appearance, in portions of the hyaline cartilage, of the large-celled variety with the formation of phosphoric esterase.¹ The present specimens were not tested for this, but it is difficult to escape from the histological analogy.

Some of the histological appearances in this case of enchondral metaplasia are consistent with Greig's² amplification of the theory or hypothesis of Leriche and Policard,³ whereby the metaplastic formation of membrane bone is believed to result from the revascularization of a region of calcium excess. According to this view, the original injury would cause tissue death and consequent calcification, and the irritation exercised by the calcium, or in this instance by repeated trauma, would bring about revascularization.

SUMMARY.

Two cases of ossification in the internal semilunar cartilage are reported, and the findings are discussed briefly: (1) From the clinical point of view; (2) From the pathological point of view, with special reference to recent work on osteogenesis.

I have to thank Mr. Elmslie for his kind permission to publish these cases and for his encouragement. The drawings and photographs are by Mr. Steward, and the histological preparations by Miss Glasscock.

REFERENCES.

- ¹ FELL, HONOR B., and ROBINSON, ROBERT. "The Growth, Development and Phosphatase Activity of Embryonic Avian Femora and Limb-buds Cultivated *in vitro*", *Biochem. Jour.*, 1929, xxiii, 767.
- ² GREGG, DAVID M., *Clinical Observations on the Surgical Pathology of Bone*, 1931. Edinburgh.
- ³ LERICHE, R., and POLICARD, A., *La Physiologie normale et pathologique de l'Os*, 1926. Paris. *The Normal and Pathological Physiology of Bone*, 1928. London.

THE FALLACY OF EXPECTANT TREATMENT IN ACUTE APPENDICITIS.

H. C. WARDLEWORTH NUTTALL.

HON. SURGEON TO OUT-PATIENTS, LIVERPOOL ROYAL INFIRMARY.

THE relative advantages of delayed and immediate operation in acute appendicitis have been so freely discussed during the past few years that the subject is becoming wearisome. In spite of this we appear to be as far from a general agreement as ever. On the whole, recent text-books favour the delayed operation.^{16, 18-22} The question cannot remain unsettled. Whether the advocates of immediate operation are justified or not, it can be shown that expectant treatment is based on error and is impracticable except in fully-staffed hospitals.

While all are agreed on immediate operation in the early or unperforated appendix, the real crux of the question is the advisability of operating on the case seen *after* forty-eight hours, with or without a palpable mass.

Table I.—MORTALITY OF OPERATION ON THE DIFFERENT DAYS OF ATTACK
(McNeill Love).

NO. OF DAYS	INFLAMMATION LIMITED TO APPENDIX		LOCAL PERITONITIS		LOCAL ABSCESS		GENERAL PERITONITIS		TOTAL		MORTALITY Per cent
	R.	D.	R.	D.	R.	D.	R.	D.	R.	D.	
1	191	1	52	1	20	0	16	1	279	3	1.1
2	264	2	61	2	45	2	69	7	439	13	2.8
3	82	2	79	12	123	8	72	19	356	41	10.3
4	21	0	67	9	71	5	26	8	185	22	10.6
5	8	0	22	2	29	0	12	5	71	7	8.9
6	10	1	31	0	32	1	9	3	82	5	5.7
7	4	0	37	1	11	0	2	1	54	2	3.6
8	7	0	19	1	9	0	1	0	36	1	2.7
9	31	0	32	1	15	0	0	0	78	1	1.2
10+	133	1	88	4	57	1	3	1	281	7	2.4
Total	751	7	488	33	412	17	210	45	1861	102	5.2

R = Recovered.

D = Died.

It is stated that "surgical interference appears to be particularly dangerous during the third to fifth days inclusive. During this period natural immunity to infection is exhausted and acquired immunity has not yet been established—that is, the patient is in a 'negative phase'."¹³ This statement appears to gain support from *Table I* published by McNeill Love,² of which he says: "This table emphasizes the accepted fact that operations from the third to the fifth day carry a high mortality. The mortality of operations on the first day is almost negligible (1.1 per cent), on the second day it is appreciable (2.8 per cent), from the third to the fifth day it is lamentable, being 10.3 per cent for this period. After the fifth day we find the mortality dwindles as the duration of history lengthens."

Table II.—COMPARISON OF MORTALITY AND AGE
(McNeill Love).

AGE	DELAYED CASES		INFLAMMATION LIMITED TO APPENDIX		LOCAL PERITONITIS		LOCAL ABSCESS		GENERAL PERITONITIS		TOTAL		MORTALITY
	R.	D.	R.	D.	R.	D.	R.	D.	R.	D.	R.	D.	Per cent
0-10	27	2	51	1	41	7	49	5	31	15	199	30	13.1
11-20	132	3	281	2	172	10	107	5	67	22	759	42	5.2
21-30	93	2	189	1	131	6	79	2	39	4	531	15	2.8
31-40	41	3	69	1	57	1	38	1	28	1	233	8	3.3
41-50	32	2	28	0	21	2	41	2	9	3	131	8	5.7
Over 50	4	0	9	1	16	2	17	2	0	2	55	7	11.3
Total	329	12	627	6	438	29	331	16	183	47	1908	110	5.4

R = Recovered. D = Died.

A very serious objection to this table is the absence of the ages of the patients. It is admitted by the advocates of the delayed operation that it is not suitable for children or the aged. Fortunately we are able to correct this table for age periods by reference to another table in the same paper giving the comparison of mortality and age (*Table II*).

It is evident that, if the majority of the patients dying of local or general peritonitis or abscess within the period 3rd to 5th day inclusive falls in the age groups 0-10, 11-20, and over 50, one would expect a much higher mortality. These figures have been surrounded by a thick line in the tables, but for the sake of greater clearness the totals are brought together in *Table III*, where it is shown that the great majority of these deaths occur in the most vulnerable age groups. It must also be borne in mind that these are the very cases which are more likely to reach this period, because diagnosis is admittedly more difficult in children and older people.

From this it follows that the argument, based on these figures, in favour of delayed operation when a patient is seen after the first forty-eight hours

is entirely fallacious. It may be noted here that my figures show no increased mortality within the 3rd to 5th day period. Nor is there, apart from these figures, any pathological evidence whatsoever that the patient is in a 'negative phase' during this period.

Table III.*

		LOCAL PERITONITIS	LOCAL ABSCESS	GENERAL PERITONITIS
Total deaths (<i>Table I</i>)	..	33	17	45
Deaths 3rd to 5th day inclusive (<i>Table I</i>)	23	13	32
Total deaths age groups 0-10, 11-20, over 50 (<i>Table II</i>)		19	12	39

* *Note*.—Fifty-five cases, including 8 deaths, are missing from *Table I*, presumably because the length of time before the onset was unknown. This does not influence the result, because whether they fall within the three-day period or not would not make any appreciable difference.

In the second place it is claimed that immediate operation is more frequently responsible for various complications and sequelæ; that drainage is required oftener, with consequent faecal fistula; and that secondary abscesses and hernia are commoner. This is certainly not true in my experience. If we are to judge from the figures published by McNeill Love,¹ drainage is required much oftener under the delayed system and complications are decidedly more frequent. In 151 cases in which operation was successfully delayed, 61 were drained and 9 suffered various complications. In 77 cases where delay failed and operation became imperative, 73 were drained and 15 suffered from complications, 5 of them fatal—a mortality of 6.5 per cent.

In a recent paper McNeill Love¹³ gives the following total mortalities:—

Immediate operation, 5.8 per cent.

Expectant treatment:—

65 per cent resolved—subsequent mortality under 1 per cent.

25 per cent forming local abscess, mortality 4.5 per cent.

10 per cent expectant treatment abandoned, mortality 6 per cent.

Average mortality with expectant treatment, under 3 per cent.

On the strength of these figures it is argued that the inherent defects of the expectant treatment may be condoned; that the extra work and mental strain thrown on all concerned are justified by better results. If this is not true—and it will be shown later that the mortality for the immediate operation in large series of cases is as low as 2.5 per cent—then it is an entirely different matter.

These disadvantages may be fairly illustrated by the description of the routine in a recent text-book¹⁸: "The delayed treatment of appendicitis *must* be carried out on the threshold of the operating theatre by the surgeon himself, for at any moment operation may become imperative." The pulse-rate is to be recorded on a special chart two-hourly, hourly, or even half-hourly. Vomiting is recorded on a vomit chart, giving the time, the quantity,

and the character. Acetone may be present in the urine after a few days' starvation, and precautions must be taken with regard to anaesthesia if operation becomes inevitable.

It is humanly impossible for the average surgeon to live constantly on the "threshold of the operating theatre". A case quoted by the same author points the moral. This case undergoing delayed treatment collapsed on the fifth day and showed signs of general peritonitis. "Within fifteen minutes gas and oxygen was administered and drainage was instituted both suprapubically and locally. His condition was critical for several days, but he recovered, and six months later appendicectomy was performed uneventfully." This case alone is sufficient to condemn the method in any hospital where a skilled surgeon and a specialist anaesthetist are not available at a moment's notice night or day.

Another serious objection is a mistaken diagnosis. The same author admits to five cases, one of them fatal, which might have been saved by immediate operation. It is also admitted that there is some wastage of hospital beds. Appendicectomy is recommended two or three months after the attack has settled down and the patient is asked to sign an agreement to that effect, otherwise there is a real danger that he may escape with his appendix! The wastage of beds cannot be dismissed lightly. Nor can the patient's point of view be disregarded. He has to face the disagreeable return to hospital later and a second absence from work that may mean the loss of a job. Finally, there is the extra strain thrown upon the surgeon and nursing staff. It should be realized that in an average ward there might be three or four of these cases under observation at one time.

I have tried to give a fair account of the expectant treatment and the objections to it. Unfortunately the same cannot be said about descriptions of the immediate operation by advocates of the delayed method. Some of them either amount to an entire misrepresentation of the facts or apply to operators who should be doing something else. Before giving the detailed figures of my own cases I shall, therefore, describe very briefly the principles which have guided me in treatment.

One of the first things to make quite clear is that 'immediate operation' does not necessarily mean a formal appendicectomy. Nothing is more certain than that anatomically and pathologically each case is an individual problem in treatment. The case with which an appendix can be delivered from the abdomen varies enormously. In an early case, with inflammation limited to the appendix and a mobile caecum, nothing can be much easier. In a late abscess, with a *congenitally* fixed caecum and the appendix possibly embedded in the retrocaecal tissues, it may be so difficult that it would be criminal to attempt it.

This is the type of case, in the hands of the inexperienced surgeon determined to remove the appendix at all costs, that has brought discredit on the immediate operation. Not satisfied with gentle separation of recent inflammatory adhesions, he sets about tearing through peritoneal layers that have been there since birth, opening up retroperitoneal cellular tissue to infection. In a mess of blood and pus the inflamed and oedematous caecum is at last

brought up sufficiently to allow removal of the appendix. If the patient does not die from toxæmia, complications are only too likely to follow.

In the great majority of cases I have employed a muscle-splitting incision (gridiron) at a site determined by the physical signs, i.e., point of maximum tenderness, palpable mass, etc. The paramedian incision is too far removed from the scene of operations, and the Battle incision, in addition to that objection, is liable to damage nerves or to lead to secondary hæmorrhage from the deep epigastric vessels later. Very gentle palpation with one finger in the peritoneal cavity is usually sufficient to determine the type of case one is dealing with.

Table IV.—MORTALITY OF OPERATION ACCORDING TO PERIOD SINCE ONSET.

DAY	I LIMITED TO APPENDIX			II LOCAL PERITONITIS			III DIFFUSE PERITONITIS			IV ABSCESS			TOTAL		
	R.	D.	Mor- tality	R.	D.	Mor- tality	R.	D.	Mor- tality	R.	D.	Mor- tality	R.	D.	Mor- tality
			Per cent			Per cent			Per cent			Per cent			Per cent
1	40	0	—	16	1	—	0	0	—	2	1	—	58	2	3.2
2	65	0	—	90	0	—	5	1	—	12	0	—	172	1	0.5
3	29	0	—	69	1	—	1	2	—	14	0	—	113	3	2.6
4	13	0	—	9	1	—	2	0	—	9	0	—	33	1	3.0
5	6	0	—	7	1	—	0	1	—	12	0	—	25	2	7.4
6	7	0	—	1	1	—	2	0	—	8	0	—	18	1	5.3
7	5	0	—	3	0	—	0	0	—	9	0	—	17	0	0.0
8	3	0	—	1	0	—	0	0	—	4	0	—	8	0	0.0
9	1	0	—	0	0	—	0	0	—	0	1	—	1	1	50.0
10+	29	0	—	4	1	—	0	0	—	14	1	—	47	2	4.0
Not stated	42	0	—	2	0	—	0	1	—	1	0	—	45	1	2.1
Total	240	0	0	202	6	2.9	10	5	33	85	3	3.4	537	14	2.5

R = Recovered. D = Died.

The appendix is removed in all cases where the general and local conditions permit of its being done without difficulty or danger; in a case of abscess no attempt is made to separate dense adhesions. In at least two cases I have closed the original wound when I have found a retrocæcal abscess densely fixed in the iliac fossa and drained it extraperitoneally through a separate incision. I submit this as a better alternative than cæliotomy (leaving a tube *down* to the abscess but not opening it) or waiting, as suggested in one text-book, till it reaches the anterior abdominal wall! On

occasion I have utilized the method of 'retrograde' removal, or in other cases the mucous membrane has been shelled out of an appendix buried in the wall of the cæcum.

As often as possible the peritoneal cavity has been closed after gently mopping out small quantities of pus. If the wall of the cæcum is much inflamed or there is a massive infection of the peritoneum, a split rubber tube or tubes is left in. Lumbar or suprapubic drainage is provided as required. In all cases where there is any chance of the abdominal wall having been infected the superficial wound is drained by a rubber dam.

Operating on these lines, a surgeon will not do any harm and he will have the great advantage of knowing exactly what type of appendix he is dealing with. In those cases where he decides not to attempt to remove it he can wait with equanimity for it to 'settle down', secure in the knowledge that there is no danger of perforation of an abscess that is draining freely. In two or three weeks' time he will be able to complete the treatment by an appendicectomy, which will then present no difficulty.

My figures are based on 551 consecutive cases operated upon during the past ten years. For the sake of comparison with *Table I*, I have arranged the figures according to the period since the onset in four groups (*Table IV*).

Apart from the fact that 10 out of the 14 deaths occurred during the first six days, there is nothing significant from that point of view. This is clearly seen in the individual groups—i.e., there is no special relation between the percentage mortality and the period since onset. On the other hand, it is interesting to observe that cases operated upon during the first twenty-four hours give a mortality of 3.3 per cent.

The cases in Group I were all acute or subacute—i.e., definite inflammation was present, ranging from catarrhal inflammation to gangrene of the mucous membrane.

In Group II a large number were completely gangrenous, and in all cases there was a definite localized peritonitis. In many cases the peritonitis occupied the greater part of the right side of the abdomen or the pelvis.

The death-rate in Group III is very high, and unless cases are operated upon earlier, probably will remain so. Of the 5 deaths one patient was aged 6 and two others were over 50 and were moribund on admission.

Group IV is particularly interesting because it shows there is no special danger in operating upon a case of abscess at any period after the onset, in spite of statements to the contrary. *Table V* gives the details of operation in this group.

Table V.—LOCALIZED ABSCESS.

	Cases	Deaths
Appendicectomy and no drainage ..	29	0
Appendicectomy and tube drainage ..	46	2
Drainage only	13	1
	<hr/> 88	<hr/> 3

Table VI shows the tendency to an increase in the number of females, noted during the past few years, and the increase in the mortality at the extremes of age.

Table VI. AGE, SEX, AND MORTALITY.

AGE GROUPS	MALE		FEMALE		TOTAL		MORTALITY
	R.	D.	R.	D.	R.	D.	
0-9	17	1	7	1	24	2	8
10-19	89	3	74	2	163	5	3
20-29	94	2	91	0	185	2	1
30-39	39	0	41	0	80	0	0
40-49	16	2	28	1	44	3	6.8
50 +	16	0	18	2	34	2	6
	279		265		530		14

Note.—Seven cases age not stated.

Table VII gives the average stay in hospital; as times goes on the tendency is to get patients up earlier, and in the uncomplicated cases the period is now approaching six days rather than nine and a half. In other groups it is probable that one cannot reduce the *average* time very much more, though individual cases may be discharged in considerably less time.

Table VII.—AVERAGE STAY IN HOSPITAL AFTER OPERATION.

				Days
Group I.	Limited to appendix	9.5
Group II.	Localized peritonitis	16.5
Group III.	Diffuse peritonitis	36.4
Group IV.	Localized abscess	25.2

On the whole, the immediate post-operative complications have been few. In the earlier cases before I began draining the abdominal wall a considerable number suppurated and sutures were cut in the ward. I am not including these or cases of slight bronchitis. All patients requiring further operative treatment or cases of definite bronchopneumonia are included. Excluding the fatal cases the complications were as shown in Table VIII.

Table VIII.—POST-OPERATIVE COMPLICATIONS.

Group I.—

(Limited to appendix):—

Abdominal wall abscess	1
Secondary drainage	1
Strangulated hernia through wound (resection of cecum, etc., primary operation)	1
Hæmophilia	1
Bronchopneumonia	3

Group II—

(Local peritonitis):—

Abdominal wall abscess	1
Secondary drainage	2
Secondary suture of wound	2
Residual abscess (one of these associated with tuberculous gland)	3

Group III—

(Diffuse peritonitis):—

Abortion and residual abscess	1
Ileus-enterostomy	1

Group IV—

(Local abscess):—

Abdominal wall abscess	1
Abdominal wall abscess with subphrenic abscess	1
Secondary drainage	1
Residual abscess	1
Ileus-enterostomy	1

Total 22

Allowing for the comparatively small number of cases, the figures compare very favourably with any cases treated on delayed lines. The total percentage mortality is small enough to allow of an increase of at least 50 per cent in a larger series and yet remain a reasonable figure.

In order to show that these figures are by no means exceptional for the immediate operation, I have collected the figures from four other papers published recently (*Table IX*):—

Table IX.

AUTHOR	TOTAL	RECOVERED	DIED	MORTALITY
				Per cent
*Raw (1930) ⁴	619	603	16	2.58
Stoney (1932) ⁷	274	267	7	2.55
†Black (1932) ⁸	340	326	14	4.10
‡Hawe (1933) ⁹	250	240	10	4.00
Nuttall	551	537	14	2.54
Totals	2034	1973	61	3.00

* Includes his *acute* cases only. † Includes 53 cases of diffuse peritonitis with 10 deaths.

‡ Includes 86 cases under ten years with 3 deaths, and 20 cases over fifty years with 4 deaths.

It might be objected that these figures cannot be fairly compared with those for the delayed method because they include cases operated upon during the first twenty-four hours. Actually if these cases, a comparatively small number, were deducted, it would not appreciably affect the percentage mortality. In my own cases, as seen in *Table IV*, the advantage would lie with those operated upon *after* the first twenty-four hours.

When the statistics in support of these opposing views are fairly considered; when it is remembered that many cases fail to subside and that the operative risk is then more than doubled; that, even if they do, they remain for several days objects of excessive care and anxiety to the nursing and surgical staffs, it is amazing that the expectant treatment has gained so much support.

In view of what has been said, there is in reality no justification for the assumption that the delayed operation is the last word in the treatment of cases of acute appendicitis seen two or three days after the onset. To many surgeons this is a retrograde step, and it is a matter of considerable importance that this should be realized by students and practitioners.

SUMMARY.

1. It is generally admitted that delayed operation is not suitable for children or the aged.

2. The expectant treatment is mainly based on figures which appear to show that operation is particularly dangerous during the period 3rd to 5th day inclusive, and it is suggested that the patient is then in a 'negative phase'.

3. It is shown that if these figures are corrected for age groups, the great majority of the deaths in that period occur in children or the aged. There is no pathological evidence for a 'negative phase' at all.

4. It is claimed that the occurrence of complications is less, and the stay in hospital considerably shorter, with immediate operation.

5. An outline of the expectant routine is given, exposing its serious disadvantages, illustrated in several instances by examples from accounts by advocates of the method.

6. The principles of the immediate operation have been grossly misrepresented in some papers. A brief description of the writer's views is therefore given.

7. This is followed by an analysis of 551 consecutive cases operated upon by the writer with a total mortality of 2.5 per cent.

8. A further series of totals is quoted from four recent papers in order to show that these results are not exceptional.

REFERENCES.

- ¹ LOVE, R. J. McN., *Brit. Jour. Surg.*, 1922-3, x, 520.
- ² LOVE, R. J. McN., *Ibid.*, 1924-5, xii, 232.
- ³ ADAMS, J. E., *Brit. Med. Jour.*, 1925, i, 723.
- ⁴ RAW, S., *Ibid.*, 1930, i, 483.
- ⁵ WILKIE, D. P. D., *Ibid.*, 1931, i, 253.
- ⁶ Roy. Soc. Med. Discussion, *Ibid.*, 1932, ii, 880.
- ⁷ STONEY, R. A., *Ibid.*, 923.
- ⁸ BLACK, J. M., *Ibid.*, 1136.
- ⁹ HAWE, P., *Liverpool Med.-Chir. Jour.*, 1933, i, 89.
- ¹⁰ RYLE, J. A., *Clinical Jour.*, 1933, Feb., 45.
- ¹¹ RAYNER, H. H., *Ibid.*, March, 89.
- ¹² BROWN, H. H., *Ibid.*, June, 247.
- ¹³ LOVE, R. J. McN., *Lancet*, 1933, i, 1229.
- ¹⁴ RAMSAY, R. A., *Ibid.*, 1317.
- ¹⁵ LOVE, R. J. McN., *Ibid.*, 1364.
- ¹⁶ QUERVAIN, F. de, *Clinical Surgical Diagnosis*, 1926, 416.
- ¹⁷ OGILVIE, W. H., *Recent Advances in Surgery*, 1928, 289.
- ¹⁸ BAILEY, HAMILTON, *Emergency Surgery*, 1930, 40.
- ¹⁹ WAKELEY, C. P. G., and HUNTER, J. B., *Rose and Carless' Surgery*, 1930, 1190.
- ²⁰ ROMANIS, W. H. C., and MITCHNER, P. H., *Text-book of Surgery*, 1930, ii, 694.
- ²¹ HOMANS, J., *Text-book of Surgery*, 1931, 963.
- ²² MAYBURY, B. C., and WILLIAMS, B. W., *Choyce's System of Surgery*, 1932, ii, 431.

THE PATHOLOGY, DIAGNOSIS, AND TREATMENT OF CONGENITAL DIAPHRAGMATIC HERNIA IN INFANTS.

By N. R. BARRETT,

FIRST ASSISTANT, SURGICAL UNIT, ST. THOMAS'S HOSPITAL;

AND C. E. W. WHEATON,

REGISTRAR, CHILDREN'S DEPARTMENT, ST. THOMAS'S HOSPITAL.

DIAPHRAGMATIC hernia can no longer be regarded as a pathological curiosity since more than a thousand cases have been recorded in detail and many hundreds have been subjected to operation. The acquired type of hernia is much more common than the congenital, and in this the general lines of diagnosis and treatment have been studied and agreed upon. The object of this paper is to record two cases of congenital hernia in infants and to remark upon the surgical treatment which is advisable at this period of life.

DEVELOPMENT.

The pleural cavities have been described by Keith as extruded portions of the cœlom, and in the early embryo they communicate freely with the peritoneal cavity. In the adult the muscular sheet of the diaphragm serves to separate the one from the other, and in view of the complexity of its development it is not surprising to find that congenital abnormalities are occasionally present. The ventral part of the diaphragm is formed from the septum transversum, which in the very early embryo is a partition lying between the heart and the abdominal viscera and growing from the splanchnic mesoderm lateral to the foregut. This septum, which derives its nerve-supply from the 3rd and 4th cervical nerves, is at first situated in the neck; as the embryo enlarges, however, it moves caudally, until at the end of the third week it occupies its final position opposite the level of the 12th rib. In this place it is joined by a thickening of the mesodermal cells at the upper end of the dorsal mesentery which bridge the gap posteriorly and fuse with the septum transversum, thus forming a continuous partition between the dorsal and ventral parts. A communication, however, remains for some time between the pleural and peritoneal cavities; it lies in relation to the 12th rib posteriorly on each side and is referred to as the pleuro-peritoneal canal; through this the pleura and peritoneum are continuous as one membrane. At the end of the fourth week the canal is closed by a double fold of serous membrane derived on the thoracic side from the pleura and inferiorly from the peritoneum. A dissection of the diaphragm in the newly born child shows that the site of the pleuro-peritoneal canals is still marked on each side by a sheet of fibrous tissue devoid of muscle fibres. The lateral portions are said to grow from the chest wall. This brief description of the

development of the diaphragm serves to illustrate the embryological significance of the various types of congenital hernia which may be found.

TYPES OF CONGENITAL HERNIA.

The classification quoted here is that of Hume, who has divided congenital hernias into the following groups: -

1. *Hernia through the dome of the diaphragm.* In its mildest form this is not really a hernia at all: it is merely a bulging of a part of the diaphragm and is known as 'eventration'. In the more advanced cases the state of affairs is different, for here there is an abnormality in the development of the septum transversum and some of the abdominal viscera may be included in the septum: the condition is nearly always associated with incomplete rotation of the gut, and the prognosis as regards life is poor.

2. *Hernia in relation to the œsophageal opening.* This is probably the commonest of all varieties, and the sequence of events which bring it about has been described by Keith as follows: "The stomach is originally almost cervical in position, but as the pleural diverticula expand and the diaphragm descends the œsophagus becomes elongated and thus the stomach also descends, maintaining its position below the diaphragm. If the œsophagus does not elongate the descent of the stomach is arrested and thus we find it placed within the posterior mediastinum surrounded by its sac of peritoneum. The sac is not formed by the protrusion of the organ but by the downward movement of the diaphragm over the stomach which is stationary." The condition has also been described in detail by Lawford Knaggs, who records the complications associated with it and shows that, although the stomach is often the only part of the intestine to be herniated, the colon, spleen, pancreas, and liver are commonly present as well.

3. *Complete absence of one or both halves of the diaphragm, allowing the peritoneum to communicate freely with the pleural cavities.* This type of hernia is incompatible with life in the majority of cases, although one patient is described as having lived till the age of 17.

4. *Hernia through the pleuro-peritoneal canal on one or other side.* The cases about to be described are instances of this type. The hernial orifice is present much more frequently on the left side than on the right and is situated in the extreme posterior part of the diaphragm in front of the 12th rib and immediately above the kidney and suprarenal body. The deficiency is usually much larger on the left than on the right where the diaphragm is supported by the mass of the liver.

5. *Hernia through one of the vascular or nervous foramina.* The commonest is perhaps the hernia in relation to the superior epigastric vessels, although on rare occasions a gap has been found in relation to the 7th costal cartilage. The developmental significance of the latter is not known.

PATHOLOGY.

It is interesting to note that the incidence of the different types of hernia varies considerably in the series reported by Hume and that reported by Keith, for in 35 cases investigated by the former there was only 1 hernia

through the pleuro-peritoneal canal, whereas in the 34 cases reported by Keith there were no fewer than 21 of this type. The explanation presumably lies in the fact that the cases collected by Keith were taken from the various London museums, whereas the other series represents an analysis of clinical material. Nevertheless the figures of Keith emphasize the fact that cases of hernia through the pleuro-peritoneal canal are not as rare as might be expected, and further that some of these live long enough to make surgical repair a possibility; indeed, he concludes his paper by saying that "from an examination of these figures I am of the opinion that a considerable number of these cases might be successfully operated upon if the condition were recognized at the time of birth." As regards this particular type of hernia, the figures he quotes are as follows:—

Hernia through the pleuro-peritoneal canal on the left side (17 cases)	{	12 died at or before birth
		1 lived six hours
		1 lived six weeks
		1 lived three months
		1 lived thirty-four years
		1 lived to old age
Hernia through the pleuro-peritoneal canal on the right side (4 cases)	{	2 died at or before birth
		2 died a few hours after birth

Further, the fact that active treatment is needed in selected cases is shown by the work of Hedblom, who found that in a series of 210 cases of congenital hernia under the age of one 75 per cent died before the end of the first month.

In those patients who survive, the onset of symptoms is usually associated with the development of intestinal obstruction. It is a matter for dispute at what stage the intestines pass into the thorax, and although it is probable that this event occurs during intra-uterine life, there are undoubtedly instances in which it occurs shortly before the onset of acute symptoms. The case recorded by Barford is an illustration of this. He attended the birth of a child which at first appeared to be normal in every way and breathed strongly. After twenty-four hours the respirations began to be laboured and the child was cyanosed. It died shortly afterwards, and at the autopsy a diaphragmatic hernia was discovered, the orifice being in the position of the pleuro-peritoneal canal; the thorax was filled with intestines. An examination of the lungs showed that both sides had expanded normally but that the left lung was compressed and airless. This suggests that at birth very little, if any, of the intestines had passed through the orifice, but that as the child cried and breathed, the coils were sucked into the thorax until the pressure upon the lungs was such that life was no longer possible.

Once the intestines have passed into the thorax the negative pressure tends to prevent or hinder spontaneous reduction such as might occur under the influence of gravity. In a number of cases, however, the amount of bowel which has herniated is not in itself sufficient to influence the workings of the chest. X-ray photographs have often shown that the mediastinum and heart may be displaced to the other side of the chest without any alarming results. The trouble begins for the patient when, owing to an overloaded bowel or tightness of the hernial orifice, which presumably does not increase much in size as the infant grows, a state of gradually increasing distension

of the gut occurs. This distension is followed by an upset in the peristalsis of the herniated bowel and paralytic ileus ensues. Owing to the absence of adhesions which is characteristic of congenital hernia here as elsewhere, strangulation is a late result, and in the average case there are several days in which the paralytic ileus is gradually developing, during which a diagnosis and treatment may be attempted; a longer delay leads to an increase of intrathoracic pressure which impedes the action of the heart and lungs and results in gangrene of the bowel.

Even in patients who have safely survived the early years of life adhesions in the thorax are very slow to form, and a review of the literature shows that when present they are most likely to involve the omentum as it passes through the orifice. In the terminal stages of the condition, and particularly where strangulation has occurred, a pleural effusion may develop.

Finally, the condition of the bowel within the abdomen must be remembered, for the afferent loop, be it stomach or intestine, may be in a more dangerous state than the herniated bowel.

The cases which have been recorded show that it is most unusual for other congenital deformities, apart from mal-rotation of the gut, to be present as complicating factors.

DIAGNOSIS.

Bradley, quoting figures from the Mayo Clinic, gives the frequency of congenital hernia as 1 in 18,000 admissions, and, from a review of this and other literature, states that it is twelve times as common on the left side as on the right. It is well known that there may be no signs of hernia during childhood, and cases such as that of Lewald show that it is even compatible with active physical exercise. Lewald's patient actually won a five-mile race. The cases, however, to which we particularly refer are those showing few physical signs at birth, but developing more serious manifestations during early infancy. There is no doubt that in many the diagnosis is extremely difficult, and Truesdale has remarked that it is more difficult than operative cure.

The cases are most commonly mistaken for congenital heart disease, in that the state presented in left-sided hernia is one of dextrocardia with attacks of cyanosis and dyspnoea. In others the sudden onset of cyanosis and loss of consciousness has presented the appearance of a cerebral hæmorrhage or pneumonia, while in the third group, when vomiting has been the predominant sign, a diagnosis of gastro-enteritis has been made.

It is therefore impossible to make a diagnosis from the history alone, but once the hernia has begun to make itself felt the symptoms are usually progressive, and repeated examinations of the thorax and abdomen will eventually establish the true state of affairs, especially in cases of hernia through the pleuro-peritoneal canal, where it is usual for a considerable amount of the abdominal contents to pass into the thorax. In this type of case Jewsbury is of the opinion that diagnosis should not be difficult.

The cardinal signs are displacement of the heart and mediastinum, associated with evidence of the presence of bowel in the thoracic cavity. The

latter should be fairly easy to detect on repeated examination, for bowel sounds will be heard in the thorax, whilst absence of intestines in the abdominal cavity will be suggested by the scaphoid outline of the abdominal wall. It remains to be said that any suspicious case should be completely investigated by radiological methods.

CASE REPORTS.

Case 1.—A male child aged 3½ months, the second child of healthy parents—another child, 4 years old, being well—was brought to hospital on account of dyspnoea since birth.



FIG. 288.



FIG. 289.

FIG. 288.—*Case 1.* X-ray of the thorax and abdomen showing the thorax to be full of gas shadows on the right side.

FIG. 289.—*Case 1.* Antero-posterior view, after a barium meal, showing that the right side of the chest is full of distended bowel.



FIG. 290.

FIG. 290.—*Case 1.* Lateral view after a barium meal, showing the position of the hernial orifice to be in the region of the pleuro-peritoneal canal.

The labour had been at full term and the birth weight was 8½ lb. The child had been breast-fed for nine weeks and after that had been given Nestlé's milk, Peptolax, and finally a Cow and Gate mixture, the changes being made on account of the slow gain in weight. At birth a mild generalized cyanosis had been noted; but, although the feet and hands had occasionally been blue, there had been no definite cyanotic attacks. The only symptoms which the parents had noticed were continuous dyspnoea and rapid respirations.

ON EXAMINATION (*Figs. 288-290*).—Temperature 98°, pulse 160, respirations 56, weight 10 lb. 7 oz. A thin cheerful child with slightly laboured and rapid respirations. Chest: movements equal on both sides; some impairment of percussion note on the right side, especially at the back. No breath-sounds were audible over the lower part of the right side of the chest posteriorly, and there were no adventitious sounds. Vocal resonance absent at the right base. Air entry poor on the right side at the apex. Heart: normal. Apex beat ½ in. to the left of the left nipple line. Abdomen: concave. Respiratory movements exaggerated.

At a later examination intestinal sounds were clearly heard over the right side of the chest. Further, it was noticed that there were two types of dyspnoea, the first occurring towards the end of a large feed, when the child became pale and collapsed and the pulse was extremely poor; the second occurring about twenty minutes after a small feed, when similar signs developed.

An X-ray examination after a barium meal had been taken showed that the small bowel had passed up behind the liver through a deficiency at the back of the diaphragm. The right side of the thorax was completely filled.

It was concluded at the time that in view of the serious general state of the child no operative treatment would be of any value and it was sent home, where it died a few weeks later. No post-mortem was available. If operation had been undertaken, it might have been possible to reduce the herniated bowel by traction from below, and if the liver could not have been fixed over the orifice, this could then have been closed by a limited thoracotomy. In view, however, of the end-result, we feel that operation should have been tried.

Case 2.—A child aged 4½ months, the only child of healthy parents. At birth the weight was 8½ lb. and progress had been normal until fourteen days before admission to hospital, when occasional vomiting had started. A letter from the doctor states that about one week before admission he had treated the child for an attack of gastro-enteritis. This cleared up quite rapidly, but it was during this time that the first attack of cyanosis was noticed. The parents had also noticed that the baby was very constipated. It was brought to hospital as a case of dextrocardia.

ON EXAMINATION.—Temperature 96°, pulse 150, respirations 40, weight 10 lb. 7 oz. Chest: impaired movement and impaired percussion note with absent air entry anteriorly on the left side; hyper-resonant note at the left base posteriorly; on one occasion intestinal sounds were heard over the upper part of the left chest. Heart: the cardiac impulse was prominent below the right costal margin, and percussion showed that the left border was at the right edge of the sternum. The heart-sounds were normal. Abdomen: the abdominal wall was retracted, but no other abnormality was found. A diagnosis of diaphragmatic hernia was made in the Out-patient Department on these physical signs.

The child took his feeds well—6 fluid ounces of Cow and Gate mixture four-hourly, although he usually had difficulty in finishing a meal; about ninety minutes to two hours later he became cyanosed and extremely distressed and the respiratory rate increased from a normal 40 to 50 to about 80 per minute. X-ray examination before and after a barium meal showed that the left side of the chest was filled with bowel, chiefly small intestine. (*Figs. 291, 292.*)

April 28, 1933.—The general condition of the child was becoming steadily worse; there was a progressive loss of weight and the attacks of cyanosis were frequent and prolonged. It was therefore decided that the only chance of saving the life of the child was by operation. It was given a course of subcutaneous saline and the stomach was washed out just before it was taken to the operating theatre.

FIRST OPERATION (Mr. Max Page).—Anæsthetic (Dr. Mennell): open ether. A left paramedian incision extending from the costal margin to the umbilicus was made. The abdomen appeared practically empty and all the intestines except the stomach had passed into the thorax. The spleen was still in the abdomen. The



FIG. 291.—*Case 2.* X-ray photograph taken before operation showing the left side of the chest to be full of gas shadows, and the heart displaced completely over to the right.



FIG. 292.—*Case 2.* X-ray photograph after a barium meal. The majority of the intestines are in the thorax and the orifice is on the left side of the diaphragm.

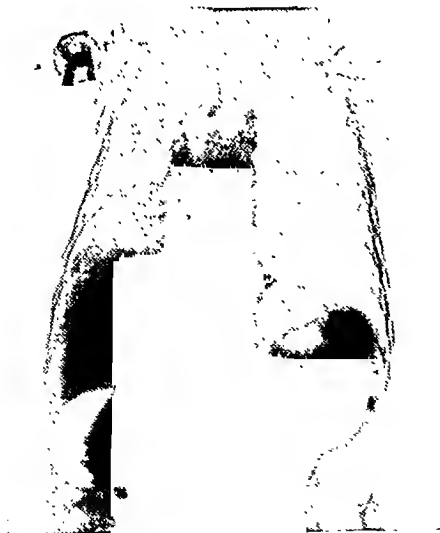


FIG. 293.—*Case 2.* X-ray photograph—taken after operation—immediately after a barium meal, showing the hernia to have been reduced and the chest free from gas shadows. The heart has already moved to the left.



FIG. 294.—*Case 2.* Showing the state of affairs one hour after that shown in Fig. 293. The heart and mediastinum have almost returned to the normal position.

herniated bowel was replaced by gentle traction from below, first the small intestine, then the ileocecal region, and finally the ascending and transverse colon were reduced from the thorax. These were packed off so that the orifice in the region of the left pleuro-peritoneal canal was easily seen; four stitches of chromic gut were used to suture the fascia on the upper part of the kidney to the anterior lip of the orifice; the abdomen was closed in layers. During the course of the operation the condition of the child improved enormously immediately the intestines had been withdrawn from the thorax. On returning to the ward the general state was good



FIG. 295.—*Case 2.* Photograph of the left half of the diaphragm from above. The pericardium with the phrenic nerve running across it can be seen anteriorly; in the middle is the lower end of the oesophagus, and posteriorly the hernial orifice. The closing sutures have been taken out.

and the child was fed with small quantities of a milk from a spoon. Later on the feeds were gradually increased till he was having eight three-hourly feeds daily. There were no further attacks of cyanosis.

SUBSEQUENT PROGRESS.—

April 30, 1933.—The bowels were open normally.

May 2.—An X-ray photograph was taken which showed that the diaphragmatic hernia had been completely reduced. (*Figs. 293, 294.*)

May 6.—Breath-sounds were heard over the upper part of the left chest, and the left border of the heart had moved to a point 1 in. internal to the left nipple line. A

further barium examination was made and was reported upon as follows: "No evidence of any hernia into the chest; bowel function normal."

May 7.—The child's condition was not so good; he was fretful and had vomited.

May 8.—Vomiting had become frequent and was associated with abdominal distension. Marked peristalsis could be seen. The increased intra-abdominal pressure had caused a large left inguinal hernia to appear. It was thought that this might account for the distension by causing some degree of obstruction, but the hernia was easily reducible and could be controlled by a woollen truss.

May 9.—General condition worse; absolute constipation for two days. A diagnosis of intestinal obstruction was made.

SECOND OPERATION (N. R. B.).—Anæsthetic: open ether. A small muscle-splitting incision was made in the right iliac fossa. Large distended coils of small bowel presented and a blind ileostomy was performed. A large amount of intestinal contents were immediately evacuated. After the operation the ileostomy functioned well, but the child became progressively weaker and died on May 16.

AUTOPSY.—At the post-mortem examination it was found that the orifice in the diaphragm had been successfully closed and that healing was well advanced. (*Fig. 295.*) The mobility of the intestines, however, was such that a portion of the small bowel at the lower end of the ileum had undergone rotation about an adhesion which had formed in the left upper quadrant of the abdomen and a length of about 2 in. had become gangrenous. From this segment of the intestine an infection had occurred and signs of early spreading peritonitis were apparent. The ileocecal region was still lying in the neighbourhood of the closed hernial orifice. The ileostomy had relieved the distension above the obstruction.

In the thorax the heart had moved over to the left side, and the left lung had expanded almost to a normal degree. There was no pleural effusion. No other congenital abnormalities were found.

TREATMENT.

In the past congenital diaphragmatic hernia has been regarded as a hopeless surgical proposition and treatment has been palliative. Indeed Hume concludes that "surgical treatment is unlikely to be required for hernia through the hiatus pleuro-peritonealis. In those cases that survive more than a few days the opening is large enough to prevent obstruction. Under certain circumstances such a condition might demand a herniotomy, but no attempt would be made to transfer the intestines to the abdominal cavity." It is now generally agreed, however, that an infant showing signs and symptoms referable to a diaphragmatic hernia can sometimes be cured if active surgical measures are possible. From a study of the various types of hernia which occur it will be apparent that some are hopeless from the beginning—for instance, a large defect cannot possibly be closed, whilst a hernia on the right side will be much more difficult to approach and reduce than a corresponding condition on the left. In the case, however, of a hernia through the pleuro-peritoneal canal on the left side, or the œsophageal opening, there is a good chance of success, and an attempt to repair the defect must certainly be made as soon as the diagnosis is established.

It will be apparent that an exact knowledge of the position of the hernial orifice will be necessary before any operation is decided upon, for on this knowledge the type of treatment depends and the relative chances of success are based. The information can be obtained in most cases from a series of X-ray photographs taken after the child has swallowed a barium emulsion. A lateral view is often helpful in deciding the position of the hernia with

reference to the front or back of the diaphragm. The improvements in diagnosis which have resulted from the use of X-rays have been clearly shown by Greenwald and Steiner.

Unfortunately it is difficult to form an idea of the size of the hernial orifice from an X-ray photograph, for it has been repeatedly shown that the amount of intestine which has passed through bears no relation to the lumen of the aperture, and this is not really surprising when it is remembered that the spleen, pancreas, or liver may have lodged in the fairway as well. It is, however, reasonable to surmise that a hernia through a patent pleuro-peritoneal canal will have a small orifice, another through the lateral sector in the vicinity of the 7th costal cartilage a slightly larger one, and those through the dome of the diaphragm a larger deficiency still. It is impossible to estimate the extent of the œsophageal herniæ.

The pessimism as to results of surgical attack upon these herniæ occurring in infants was based upon the fact that very few cases had been subjected to operation and the methods which had been found of value in traumatic and adult cases were used without success. In the last ten years, however, a considerable number of babies have been operated upon and the results have been duly recorded, so that it is possible to form an opinion about the relative merits of the various techniques which have been advocated by different surgeons.

It must be emphasized at the outset that the problem is quite different in infants and in those who survive childhood without symptoms, for in the latter the state of acute emergency has seldom been reached, a set operation can be planned, and the whole course of treatment worked out according to schedule. Further, the size of the affected parts and their relative strength and mobility are considerably changed as the patient grows older, so that a method of approach which is suitable for the one will be of no avail for the other.

The surgeons who have contributed most to our knowledge of treatment of these herniæ in the adult are Hedblom and Truesdale. The former has recorded a series of over 1000 cases of diaphragmatic hernia of all kinds, in which operation was carried out in nearly 400; it is significant, however, that in this enormous series he mentions only 6 cases under the age of 5, which had been reported before 1924. Since this time a fair number of operations have been successfully done in infants, and the results of the treatment in some will be referred to and criticized.

Pre-operative Treatment.—As regards pre-operative treatment, it is important to improve the general condition of the patient as much as possible, and this can be achieved to some extent by subcutaneous, intravenous, or rectal salines; again, it is essential that the stomach be emptied by washing it out before the operation starts, for it will probably be dilated and may be full of fluid. In the case which was operated upon by Leopold the stomach was greatly distended with fluid, which not only made the approach to the hernial orifice difficult, but played an important part in the death of the child, for during the manipulations necessary to reduce the hernia the contents of the stomach regurgitated into the pharynx and caused asphyxia, so that the operation had to be temporarily abandoned, and the child eventually died

of shock, although the defect in relation to the œsophagus had been successfully repaired.

Anæsthesia.—It would appear that the anæsthetic of choice has not yet been found, for almost all the surgeons about to be referred to have selected a different method. It has been shown that a sac is present in less than a quarter of the cases of congenital hernia, and therefore in the majority a pneumothorax will be produced whether the abdominal or the thoracic approach is chosen. This has led to the use of positive-pressure anæsthesia, and Truesdale attributes his successes to a large extent to this fact. On the other hand, it has been pointed out that if a laparotomy is done, the pneumothorax will not be a serious matter, for the lung and mediastinum on the affected side are already displaced and the size of the hernial orifice is not as large in a favourable case as the wound made by thoracotomy. At the other extreme is a local anæsthetic such as was employed in the operation successfully accomplished by Iizuka and Sano, but the time taken in administering the anæsthetic and in keeping the child still must be considerable and must add to the risk of an already serious procedure. There remains to be considered the use of open ether, and to the writers this appears to be best. It is certainly an anæsthetic which can be tolerated with reasonable safety by infants in a dehydrated state, a fact which has been well shown in the cases of congenital pyloric stenosis which have been operated upon by Max Page with such good results. It would not, however, be suitable for an extensive thoracotomy.

Method of Approach.—The method of approach is a much debated point, and before discussing the relative merits of laparotomy and thoracotomy it is well to point out certain statistics. In the first place the general mortality of all cases which have been operated upon, according to one authority, is 54.5 per cent, whereas in infants it rises to 80 per cent. In the absence of the signs of intestinal obstruction the mortality for all cases is as low as 23.8 per cent. These figures show clearly that the advice which used to be given—namely, do not operate unless in the presence of obstruction or thoracic compression—is responsible to a great extent for the high death-rate, and we consider that every case in which a reasonable chance of success is present should be dealt with as soon as the diagnosis is made.

Thoracotomy.—The advocates of thoracotomy as a method of approach maintain that the exposure obtained by spreading the ribs is very much better than that by laparotomy, and this is especially true in the herniæ through the pleuro-peritoneal canal and the lateral part of the septum, which, after all, are the most amenable to treatment. In addition to this, once the hernia has been reduced, it is much easier to close the hole from above than from below, for not only is the field free from the coils of distended intestine, but if any difficulty is experienced in bringing the edges together, the phrenic nerve, which is lying close by upon the pericardium, can be put out of action and the corresponding half of the diaphragm relaxed. If it is still difficult to close the orifice, the lower two ribs can easily be severed, a method which was used by Bettman and Hess in the operation successfully performed by them.

Laparotomy.—In spite of these advantages, the safest and best approach in infants is by laparotomy. In the first place those cases in which a hernial

sac is present, as is often the case in the œsophageal variety, will not suffer the additional embarrassment of a pneumothorax however small this may be. Then again the general advice of Hedblom may be taken to refer to this particular type; he writes: "Laparotomy and not thoracotomy irrespective of the comparative mortality of each is indicated in all cases in which obstruction is present, because the state of affairs cannot be accurately diagnosed until it has been seen, and because the patient's best chance of recovery may lie in a drainage of the afferent loop." In these infants, who cannot describe or complain of their symptoms, it is almost certain that by the time a diagnosis is made some degree of paralytic ileus or obstruction will be present. Even the advocates of the thoracic approach are agreed on this point, for Truesdale has reported a case in which he did a œcostomy to overcome the acute obstruction within the abdomen and shortly afterwards repaired the hernia by thoracotomy. Apart from the signs of obstruction, however, there are many in which the correct diagnosis is not made until the abdomen has been opened; for instance, Towers has reported a case which was diagnosed as an acute intussusception, a mass being palpable in the left upper quadrant of the abdomen. Laparotomy was undertaken and the spleen was found to have undergone torsion; when this had been reduced a diaphragmatic hernia was seen through the posterior quadrant of the left side of the diaphragm. As there were no signs of obstruction at the time, the repair of the hernia was deferred and was successfully completed at a second laparotomy later on when the child had recovered from the first operation.

Reduction of the Hernia.—Finally, the question of the actual reduction of the hernia is of the greatest importance. These cases are characterized by the large amount of bowel which herniates, all of which must be handled during the process of reduction, and the shock to the patient is considerable. It is therefore a fact of great importance that, whereas it is an easy matter to pull the intestines through the orifice from the abdominal side, it is practically impossible to push them through from the thorax. In addition to this, if any of the viscera have passed through as well, the orifice may be completely occluded. This difficulty was encountered by Coryllos and Tow, who have recorded the case of a baby 13 days old in which they were able to repair the opening. The child, which was apparently normal at birth, remained well until the seventh day, when it developed a sudden severe attack of cyanosis and dyspnoea. Radiography showed a hernia through the left pleuro-peritoneal canal and operation was advised. The hernial orifice was approached by thoracotomy and the chest was found to be full of distended intestines. There were no adhesions, but reduction of the hernia was impossible because the spleen had passed through and was held firmly against the orifice by its mesentery. The thoracic incision was therefore prolonged on to the abdomen to the left of the mid-line, and reduction of the spleen and intestines was easily effected from below. The operation took forty-five minutes in all. In spite of the severity of this, the patient survived, which reminds one how much a very young baby can resist, and it would appear that not only can they resist anoxæmia to an extraordinary degree but that the heart is very strong at this age. The same difficulty of reducing the hernia from above

was experienced by Bettman and Hess in the case already mentioned, and they were forced to do a laparotomy as well. It is of interest that the hernia with which they were dealing was of the type in which the defect is laterally placed opposite the 7th costal cartilage. It has been suggested that the difficulty can be overcome by stretching the orifice manually or by extending it in a lateral direction, but even this has proved unsuccessful and must be considered a destructive procedure when the whole thing can be done from below without difficulty. Further, if by any remote chance some adhesions had already formed and were preventing reduction, a thoracotomy can still be done as a secondary measure.

It might be argued that the method of choice is a combined laparotomy and thoracotomy, but there is no doubt that in infants this should be avoided if possible. From a study of the operative mortality in older cases it is apparent that the combined operation is consistently associated with a higher mortality than either of the other two done separately. It has already been pointed out that the mortality in these infants is high in any case, and to attempt the double event when the whole procedure can be done by laparotomy alone is to make the operation an altogether more serious business. There have been a number of instances in which success has been achieved from below, and the surgeons who report these cases lay stress on the fact that the operation has not been difficult. The cases described by Schönbauer and Warkany, Donovan, McCleave, Iizuka and Sano, and Leopold should be particularly consulted. From the anatomy of the part it will be seen that the orifice of a hernia through the left pleuro-peritoneal canal can be easily closed by suturing the fascia which lies on the front and superior aspect of the kidney and suprarenal body to the peritoneum on the anterior part of the orifice. This fascia is well suited to bridge the gap because it is strong and at the same time fairly mobile on the front of the kidney.

Another practical difficulty which arises is that the distended intestines which have been reduced from the thorax may be too large to fit into the abdomen. This can be overcome by pricking the gut in several places and allowing the gas to escape, the points of puncture being invaginated in the ordinary way.

It has been noticed in several of the cases that the general condition of the patient improved as soon as the intestines had been withdrawn from the thorax; this was due to the reduction of the intrathoracic pressure and also to the fact that the compressed lung had to some extent become operative. This advantage should be pressed home at the conclusion of the operation and the child should be given CO_2 to breathe; if an intratracheal or positive-pressure anæsthetic has been used, the lung can be actively expanded. Further, by passing a pneumothorax needle into the pleural cavity on the affected side the pneumothorax can be reduced to a minimum by slowly sucking out the air. This procedure has been done successfully on several occasions, and it has been shown that the absence of adhesions allows the heart and mediastinum to move across to their normal position almost immediately. If the pneumothorax is not removed in this way, the absorption of the air takes about fourteen days, a fact which has been demonstrated by subsequent X-ray photographs.

SUMMARY.

1. A certain number of cases of congenital diaphragmatic hernia occurring in infants can be cured by surgical intervention.
2. The most favourable types are those through the pleuro-peritoneal canal on the left side, lateral defects in the septum, and small hernie in relation to the œsophagus.
3. Laparotomy rather than thoracotomy is the method of choice.
4. Open ether is the best anæsthetic.
5. The hernia can be easily reduced from below, but only with extreme difficulty from above.
6. The operation if done from below is not difficult in favourable cases.
7. Adhesions are very rarely present and need not be anticipated.
8. The pneumothorax should be removed at the conclusion of the operation.

Our thanks are due to Dr. R. C. Jewesbury and Mr. Max Page for advice and criticism and for permission to publish these cases, which were under their care. Dr. Fildes has kindly supplied the X-ray photographs.

REFERENCES.

- HUME, J. B., *Brit. Jour. Surg.*, 1922, x, 207.
 KEITH, Sir ARTHUR, *Ibid.*, 1924, xi, 455.
 KNAGGS, R. LAWFORD, *Lancet*, 1904, ii, 358.
 KEITH, Sir ARTHUR, *Brit. Med. Jour.*, 1910, ii, 1297.
 HEDBLUM, C. A., *Trans. Amer. Surg. Assoc.*, 1931, xlix, 448.
 BARFORD, L. J., *Brit. Med. Jour.*, 1932, ii, 794.
 BRADLEY, W. N., *Arch. of Pediatrics*, 1930, xlvii, 384.
 LEWALD, *Amer. Jour. Roent. Sci.*, 1925, 13.
 TRUESDALE, P. E., *Trans. Amer. Surg. Assoc.*, 1931, xlix, 114.
 GREENWALD, H. M., and STEINER, M., *Amer. Jour. Dis. Child.*, 1929, xxxviii, 361.
 LEOPOLD, J. S., *Ibid.*, 1927, xxxiii, 597.
 IZUKA, T., and SANO, V., *Orient. Jour. Dis. Infants*, 1932, xi, 23.
 BETTMAN, R. B., and HESS, J. H., *Jour. Amer. Med. Assoc.*, 1929, xcii, 2014.
 TOWERS, R. J., *Brit. Jour. Surg.*, 1927, xv, 332.
 CORYLLOS, P. N., and TOW, A., *Jour. Thoracic Surg.*, 1932, ii, 56.
 SCHONBAUER, L., and WARKANY, J., *Zeits. f. Kinderheilk.*, 1930, 1, 125.
 DONOVAN, E. J., *Surg. Clin. N. Amer.*, 1931, xi, 521.
 MCCLEAVE, T. C., *Amer. Jour. Dis. Child.*, 1917, xiii, 252.

PROSTATECTOMY WITH CLOSURE: FIVE YEARS' EXPERIENCE.

By S. HARRY HARRIS,

HONORARY UROLOGIST, LEWISHAM HOSPITAL, SYDNEY.

THE evolution of the operation of suprapubic prostatectomy with complete closure of the bladder has, in the author's hands, been a gradual process from the first stage—i.e., the re-formation of the floor of the prostatic urethra—which was described by him at the second session of the Australasian Medical Congress (British Medical Association) in Dunedin, New Zealand, on March 26, 1927.¹ In its completed form it has been practised by him, with minor variations, in both one- and two-stage operations for upwards of five years dating from October, 1927. The prostatectomy operations for simple hypertrophy performed by the author during the five-year period to October, 1932, are shown in the table below.

SUMMARY OF CASES SUBMITTED TO OPERATION FOR SIMPLE HYPERTROPHY DURING THE FIVE-YEAR PERIOD TO OCTOBER, 1932*.

TYPE OF OPERATION	NO. OF CASES	TOTALS
One-stage prostatectomy	322	371
Two-stage prostatectomy	49	
Deaths 10 = mortality rate 2·7 per cent		
Prostatectomy with closure	356	371
Prostatectomy with suprapubic drainage ..	15	
Reasons for drainage: Two-stage operation (1st stage elsewhere in 5), incomplete technique, 7 cases; urethral calibre too small to admit catheter, 5 cases; presence of foul diverticulum, with plastic on stoma, 2 cases; profuse urethritis, 1 case		
Preliminary cystostomy:—		
Died within one month	5	61
Later prostatectomy	49	
Left as permanent cystostomy	7	

Since the last paper,² presented before the American College of Surgeons on Oct. 14, 1929, re-opening of the bladder during convalescence has had to be carried out in 3 of 261 patients (1·11 per cent). Of these, one had incomplete drainage due to a faulty catheter and required cystostomy on the seventh

* In the seven-month period from October, 1932, when this paper was completed, to May, 1933 (this period having been occasioned by delays in transit), 42 additional cases have been submitted to the operation with 1 death, making the total number of deaths for the series 11 and the mortality rate slightly lower than 2·7 per cent.

day: one had a severe reactionary hemorrhage immediately after operation, for which cystostomy and blood transfusion were carried out, followed by complete recovery; and in the third patient the tip of the catheter had found its way through the bladder incision, and, although his urine was free of blood and no urinary extravasation had taken place, there was a considerable amount of suprapubic tenderness and edema, and on the seventh post-operative day the suprapubic wound was re-opened, the catheter readjusted, and the prevesical space drained—this was followed by complete recovery.

Deaths.—The number of deaths, as stated above, was 10, the causes of which were: Inanition, 47th day, one case; pneumonia, 6th day, two cases; acute intestinal obstruction, 14th day, from carcinoma of the sigmoid, one case; paralytic ileus, 12 and 14 days after spinal anaesthesia, two cases; polycythemia, pulmonary embolism, 7th day, one case; auricular fibrillation and cardiac dilatation, 4th day, one case; shock, prostatectomy plus deep diathermy to infiltrating carcinoma of bladder base, death 12 hours after operation, one case; panophthalmitis, excision of eye, death 3 days later and 15 days after prostatectomy, one case. This last patient had had a preliminary cystostomy six weeks before the prostatectomy. There had been a number of small pustules below the fistula which were slow to heal. These were in the line of the prostatectomy incision. The day before operation they were touched with pure carbolic acid, and the area of skin containing them was excised during the skin incision. His wound broke down and he developed a general pyemia. This is a valuable example of the inadvisability of ever carrying out a prostatectomy until all conditions are as reasonably favourable as they can be made. ;

REPORTS OF PROGRESS.

Papers recording progress of the operation in the author's hands have appeared at intervals during the past five years.²⁻⁴ A moving picture film of the operation was shown before the Annual Congress of the American College of Surgeons in Chicago in October, 1929. In September of that year, through the courtesy of Dr. Joseph F. McCarthy, an operative demonstration of the actual operation was given at the Post-graduate Hospital, New York, before a representative gathering of urologists.

The operation, which is slowly but surely gaining adherents both at home and abroad, is not an operation for the occasional prostatectomist, but will, the author believes, become much more widely adopted when the comparative simplicity of the various steps and the safety and soundness of the surgical principles involved become more widely appreciated.

It will be the object of this paper to present the consecutive steps of the operation in as concise and clear a way as possible; to describe the minor, though important, alterations in surgical technique which have been made since the last publication; and to detail the precautions which the author, in his practice, has found to be essential to the successful issue of any considerable consecutive series of cases where this technique is used.

The particular features of the operation which most commentators appear to miss are, briefly: (1) The complete and immediate control of

hæmorrhage by suture; (2) The plastic covering of all the raw surfaces in such a way that the prostatic urethra is in the great part re-formed and the prostatic cavity obliterated, with the result that healing takes place generally by first intention, septic complications are remarkable for their rarity, fistula formation does not occur, and post-operative recurrence of obstruction is completely obviated.

Notwithstanding 'armchair' criticism concerning the permanency of the plastic portion of the operation on the prostatic bed, extended experience, both cystoscopic and urethrographic, has proved quite definitely that the parts do remain in the position in which they are sewn at the operation. If other than ocular evidence be needed, it is to be found in the fact that when the plastic reconstruction of the floor of the prostatic urethra has been omitted, post-operative recurrence of obstruction has been a not uncommon sequel, while it has proved to be entirely absent in those cases in which this reconstruction has been carried out.

Almost without exception, surgeons who have adversely criticized this operation either have no first-hand knowledge of it, have not followed the technique in anything like its entirety, or have made such alterations as to render it unrecognizable.

There have, as with all new procedures, been several suggested modifications of the original, but, so far as the author is able to judge, none so nearly as the original reproduces the normal anatomical and physiological relationship: practically all result in the immediate formation of some type of diaphragm fitting tightly round the catheter; and none seems likely to be so free from annoying post-operative complications as the original has proved to be.

The author would appeal to all who purpose doing this operation to follow the technique in its entirety. Many minor variations have been 'tried out' by him and discarded, and the technique finally adopted has not only been evolved as the result of extended experience, but has stood the test of time. More particularly would he deprecate minor alterations in surgical technique so far as the prostatic cavity is concerned. The reconstruction of the floor of the prostatic urethra is the one vital point, or keystone, upon which a successful plastic operation of this type must be constructed. A complete, clean enucleation of the prostate is, of course, the indispensable foundation.

The results obtainable by this operation, so far as mortality and morbidity are concerned, present a marked contrast to those obtained by some other widely practised 'open' methods, as for example that of Judd and Hunt of the Mayo Clinic, or that of Thomson-Walker as described by Swift Joly and Kenneth M. Walker.⁵ Swift Joly, after making a brief and inaccurate reference to the author's operation, describes some of his own attempts to "reduce the size of the raw area in the prostatic cavity", records his failures, and adds: "The prostatic cavity always becomes infected and is unsuited for plastic operations". As a matter of actual fact, where the author's operation more or less completely covers up the raw surfaces, thus guarding them from infection and limiting absorption to the minimum, the Thomson-Walker operation, as described by Swift Joly, not only leaves the entire prostatic cavity wide open for infection and absorption, but makes it

even wider by cutting out a deep V from the apex of the trigone, thus deliberately exposing to even further infection the most vulnerable portion of the urinary tract. Is it any wonder that septic manifestations are common? This last-named step is undertaken with the avowed object of preventing post-operative recurrence of obstruction. According to the admissions of its sponsors it fails in many cases even to do this, and extensive re-operations have to be performed for the cure of post-operative fistula and of cicatricial contracture of the bladder outlet. Finally, the persistent post-prostatectomy sepsis which is featured so largely in their published post-operative results is, in Kenneth Walker's words, "sometimes so troublesome that patients have learnt to wash out their bladders daily with as little difficulty as most of us experience in cleaning our teeth".

One would have felt justified in thinking—unless, of course, one's statements were entirely disbelieved—that a welcome would be assured for a method which promised even partial immunity from the appalling list of post-operative sequelæ so frankly acknowledged by these surgeons, and a reduction of the high mortality, which is stated to be "in the region of eighteen per cent. at general hospitals and eight per cent. at special hospitals".

In addition to the reduction of the mortality and morbidity rates resulting from the author's operation, the convalescent period has been shortened and made infinitely more comfortable. The immediate and positive control of hæmorrhage by suture lifts a load from the minds alike of patient and surgeon. The absence of post-operative urinary leakage through the wound, which, though by no means invariable, has been the general rule, is a further comfort. As stated above, in the entire series of 371 cases there has been no single instance of post-operative fistula formation or of recurrence of obstruction. There has been one instance of post-operative hernia in the entire series.

In the operation practised by the author there are two factors of safety which are of paramount importance, inattention to either of which is likely to spell failure to the plastic portion of the operation however carefully this may be carried out. These are: (1) The employment of surgical asepsis and antisepsis in the care of the catheter in both the pre- and post-operative periods; and (2) The employment of the intra-urethral method of digital enucleation of the prostate.

1. The Indwelling Catheter and the Demand for Surgical Cleanliness.—The most careful aseptic and antiseptic ritual must be observed in the care of the indwelling catheter when this is employed, both in the pre- and post-operative periods. Any slovenliness in this regard, apart from any immediate ill effects, will reap its own harvest of post-operative septic sequelæ. The method, so often seen, of allowing the open end of the catheter to drain into a urinal placed between the patient's thighs is fraught with danger and should no longer be tolerated; the way is wide open for infection which is not slow to develop. Only less objectionable is allowing the end of the catheter, perhaps plugged by a wooden peg, to swing loose among the bed-clothes.

2. The Intra-urethral Method of Digital Enucleation of the Prostate.—The method of Freyer which held sway for so many years is too liable to be associated with complete removal of the verumontanum, the anterior commissure, the anterior portion of the prostatic urethra down to the triangular

ligament, and sometimes even the membranous urethra. An unnecessarily wide and particularly vulnerable area is thus opened up for infection. In the author's practice the few cases in which the verumontanum has been accidentally removed are the only ones in which a slight tendency to persistent post-operative urinary leakage has developed, and, by the same token, were the only ones which required the passage of a sound at any period during convalescence. Particularly would the author desire to utter a word of warning against the open instrumental method of carrying out Freyer's operation, as this must almost of necessity entail complete removal of the prostate, verumontanum, anterior commissure, and prostatic urethra. No method of plastic surgery, however well carried out, will overcome the above-mentioned defects in enucleation. With the method of bimanual intra-urethral enucleation employed by the author (*see below*), which is merely an elaboration of that described many years ago by Bentley Squier, of New York, the verumontanum is preserved, the anterior commissure is not disturbed, and the mucosal covering of much of the prostatic urethra remains intact (*see Fig. 299*).

SURGICAL PRINCIPLES INVOLVED AND OPERATIVE REQUIREMENTS.

The operation places suprapubic prostatectomy in line with accepted principles of general surgical practice in so far that it makes immediate provision for: (1) The positive control of hæmorrhage by suture; (2) The re-formation of the prostatic urethra; (3) The covering up of raw surfaces and obliteration of dead spaces (*viz.*, the prostatic cavity and the cave of Retzius); (4) The abolition of unnecessary drainage (*i.e.*, of suprapubic cystostomy); and (5) First-intention healing. The first three are the vital or essential principles of the operation, and they are therefore of primary importance; the complete closure, though also extremely valuable, is by comparison a secondary consideration.

The operation demands complete visual exposure of the base of the bladder, and may, if so desired, be performed through a long vertical or transverse incision, when only one special instrument—namely, the combined needle and needle-holder—will be essential; or, as the author prefers to do it, through a short transverse incision, from $2\frac{1}{2}$ to 3 in. in length, for which a set of self-retaining, electrically-lighted bladder retractors has been devised (*see below*). Any desired bladder retractors may be employed in conjunction with a larger incision, but the set illustrated (*see Fig. 297*) widely opens up the bladder base and prostatic cavity, and permits the employment of a much smaller incision without impairing the facility with which the successive steps of the operation may be carried out. When the complete closure is practised in conjunction with a long incision of the abdominal parietes and bladder, drainage of the prevesical space *must be provided*. The author's experience and voluminous correspondence have shown the very real risk of urinary extravasation when this precaution is omitted. This very serious complication has not been encountered by the author in this last five-year period, *i.e.*, during the currency of the present operation.

SPECIAL INSTRUMENTS.

The special instruments employed are four in number: they are as follows:—

1. **The Boomerang Needle-holder.**—An improved and perfected design. This needle-holder is now well known. It is a modification by the author of the well-known pattern of Young. The actual length assembled, but excluding the needle, is $9\frac{1}{4}$ in.: the over-all length, with the large-size needle mounted, 10 in. The author's large-size needle only is illustrated (*Fig. 296*), the smaller being approximately two-thirds the size. The large-size needle is indispensable for the placing of the trigonal and of the anterior sutures in the operation as now performed. A smaller needle does not take a sufficiently wide bite.

In order to avoid hæmorrhage from the needle puncture it is important that the needle should not have a terminal sharp cutting edge. The needle should be sharpened only at the actual point. It is made of rustless steel and must be kept sharp.

The sole drawback in the past to the use of this needle-holder has, in the author's experience, been the occasional 'jumping out' of the needle from the end of the needle-holder. This difficulty has now been overcome by replacing the locking pin of the original design with a small and readily adaptable double clip and pin device (*see Fig. 304*) which is positive in action and made of rustless steel. The complete instrument is made by Messrs. Elliotts and Australian Drug Limited, O'Connell Street, Sydney.

2. **Ligature Carrier for the Boomerang Needle-holder.**—The use of this carrier renders easy the placing of the suture in the needle. It has been described in previous publications.

3. **The Bladder Retractors and Self-retaining Frame.**—The set consists of three electrically-lighted retractors, two lateral and one posterior, adapted from the old handled pattern of Thomson-Walker (*Fig. 297*). The lamp for the left retractor is carried near the top of the blade, that for the right near the bottom, while for the posterior the light is set about the centre. An even diffusion of light is thus procured.

The value of this set of retractors has been greatly enhanced by the addition, since the last publication, of a self-retaining frame. This is of a very simple design and made in two parts with a 'pull-and-push' union for ease of sterilization and transport (*Fig. 297*).

The couplings for the two lateral retractors are set on the frame at an angle, so that the retractor stems run obliquely from without inwards and downwards. By this means the retractor blades are given an outward and upward cant and bear directly on the side walls of the bladder, while there is little, if any, pressure on the abdominal parietes. This ensures wide lateral retraction of the bladder base.

4. **Anterior Retractor.**—In addition to the self-retaining set, a narrow



FIG. 296.—The author's large-size boomerang needle. There is no cutting edge: the point alone is sharp. ($\times \frac{1}{2}$.)

recurved, long, anterior retractor made of spring steel is employed which dips below the anterior lip of the prostatic rim and lifts this forward, thus widely opening up the prostatic cavity and greatly facilitating the placing of the sutures (*Fig. 297, 304*).

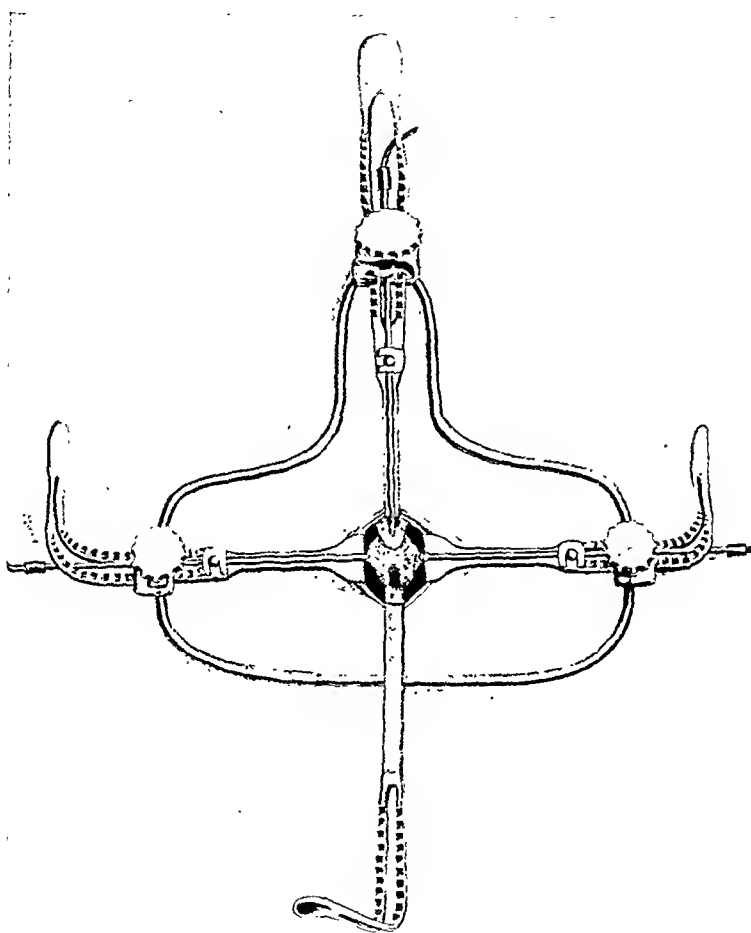


FIG. 297.—The author's electrically-lighted bladder retractors and self-retaining frame. The anterior retractor is not electrically lighted and does not connect with the frame. The complete exposure of the field of operation is well shown. The tip of the anterior retractor is seen lifting the anterior lip of the prostatic cavity forward.

PREPARATORY TREATMENT.

Preliminary catheter drainage is employed for at least ten days (or preliminary cystostomy for at least one month, in cases in which catheterization is unsuccessful) if the urine is dirty, if the residual urine exceeds 4 oz., or if the renal function is below par. Patients whose general condition is below par and who for that reason require pre-operative preparation often improve

to an extraordinary extent with a retained catheter, even when no residual urine is present. In this type as a rule, and generally in cases of any doubt, the retained catheter is employed as a preliminary, though sometimes intermittent catheterization combined with rest in bed may bring about the desired result in a more comfortable way.

Vas Ligation for the prophylaxis of epididymitis is practised as a routine. The earlier in the preparatory treatment the insertion of the sutures, the more successful will be the result. Silkworm gut subcutaneous transfixion sutures are used, and removed after seven days.

Method of Emptying an Over-distended Bladder.—Any suitable rubber or silk catheter is passed and its funnel end closed. The smallest size hypodermic needle is then plunged through the wall of the catheter into its lumen. A length of narrow rubber tubing connects the hypodermic needle to a bottle at the bedside. Not more than 4 oz. of urine per hour are allowed to flow through the needle. To ensure this it may be necessary partially to obstruct the rubber tubing. When the bladder is emptied, generally at the end of twenty-four or thirty-six hours, the needle is removed and the catheter is connected up to a bottle at the bedside as described below. This method is of proved safety and simplicity, and is very generally applicable.

Routine Adopted when a Retained Catheter is Employed.—The bladder is washed out thoroughly once or, if necessary, twice daily with a solution of permanganate of potash of pale pink colour, then with plain sterile water, and completely emptied. Four ounces of a 1-3000 solution of silver nitrate are then run into the bladder and the catheter is clamped for half an hour. When the bladder is dirty the strength of silver nitrate is increased gradually to the limit of tolerance, sometimes even up to 1-1250. Generally the dirtier the bladder the greater the tolerance for silver nitrate. The silver nitrate solution must not be strong enough to cause pain.

The catheter is drained by a glass connecting tube and a length of rubber tubing to a bottle at the bedside containing antiseptic solution, into which the tubing dips. No method of antiseptis will clean up a dirty bladder, or prevent infection of a clean one, when the end of the catheter is placed, as is so often seen, in a urinal between the patient's thighs.

The catheter is changed at least every third day, the patient, if well enough, being given a hot bath between changes of catheter. Always before insertion of the catheter the glans penis is mopped with methylated spirit and the urethra irrigated with 1-5000 solution of oxycyanide of mercury. The catheter lubricant consists of 1-500 oxycyanide of mercury cream (tragacanth-glycerin).

Urinary Antiseptics.—Sodium benzoate, 15 to 20 gr. t.d.s., and hexamine, 10 gr. t.d.s. or oftener, are given by mouth. When sodium benzoate disagrees with the patient, acid sodium phosphate may be employed instead in the same doses. This medication is continued, except for a short remission after operation, throughout the patient's stay in hospital. Confinement to bed is usual only for the first day or two of the catheter treatment.

If this technique is faithfully carried out, there will be found few even badly infected bladders which cannot very efficiently be cleaned up, and the prostatic bed may thus be made a safe place for plastic surgery.

Digitalis.—This may generally be administered with advantage. It is, however, only prescribed after complete routine examination by a physician, which is insisted upon in all cases.

Indigo-carmin Test.—After a minimum of ten days when catheter drainage is indicated, but not until such time as the patient's condition is considered otherwise favourable, an intravenous injection of 10 c.c. of a 0.4 per cent solution of indigo-carmin is given. Unless a good blue coloration (*see below*) appears in the urine within ten minutes, the patient is considered unsuitable for prostatectomy for the present, and is either further prepared by the retained catheter or submitted to preliminary cystostomy. Under no circumstances is a prostatectomy undertaken, whatever the other tests of renal function may indicate, unless the result of the indigo-carmin test is considered good enough to warrant it. This is regarded as the final renal test of operability.

The present régime carries with it a considerable improvement in the operability rate. Patients, whose renal function would previously have been considered to render prostatectomy either hazardous or even inadmissible, are now with confidence offered the operation. Where formerly a dark blue coloration of the urine was deemed necessary in the final test of operability, operation is now safely undertaken in patients whose indigo-carmin test yields a very much lighter blue. Suitable efforts will, of course, have been made to bring it up to the usual dark blue coloration.

OPERATIVE TECHNIQUE.

The steps of the operation may be summarized as follows:—

1. Complete washing and emptying of the bladder and irrigation of the urethra immediately before operation.

2. Draping of the towels on the patient in such a way that separate and individual access may be had to the rectum, penis, and abdominal incision. This method has been described in previous papers.

3. A transverse incision through the skin and fat, $2\frac{1}{2}$ to 3 in. in length and 1 in. above the level of the symphysis.

4. Incision of the bladder at the highest point, after pushing back the peritoneal reflection. All bleeding points on the cut edges of the bladder are carefully tied to ensure that there will be no post-operative bleeding from this source.

5. The enucleation of the prostate. The bimanual method of intra-urethral enucleation of the prostate is employed by the author, two fingers of the left hand, on which two gloves are worn, being passed into the rectum. With the small transverse suprapubic incision which he uses, the added assistance afforded by the fingers in the rectum is invaluable. The right forefinger is passed into the prostatic urethra and breaks its way through the mucous membrane at the antero-inferior aspect of one or other lateral lobe and just external to the verumontanum. The inferior and superior borders of the lateral lobes are followed round to the mid-line first on one side and then on the other (*Fig. 298*). At the end of the enucleation the parts are left approximately in the condition shown in *Fig. 299*. By this method the prostate

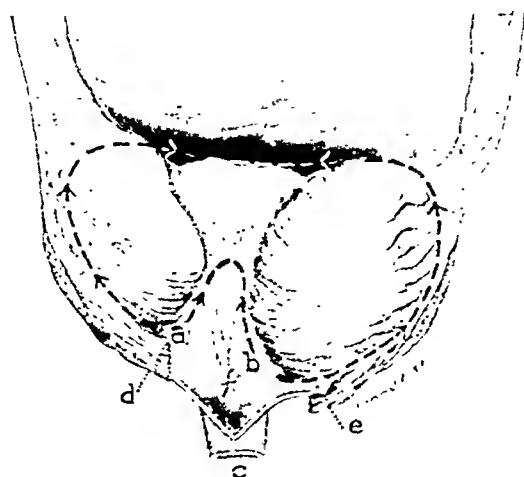


FIG. 298.—Drawing of post-mortem specimen of bladder and prostatic urethra, opened from the front, illustrating the intra-urethral method of enucleation of the prostate described in the text. The broken black line and the arrows thereon indicate the course followed by the finger during the enucleation. The points *a* and *b*, opposite the antero-inferior aspect of each lateral lobe, indicate the site at which the enucleation is begun on each side; *d* and *e*, The cut edges of the muscle of the anterior commissure; *c*, The membranous urethra; *f*, The verumontanum.

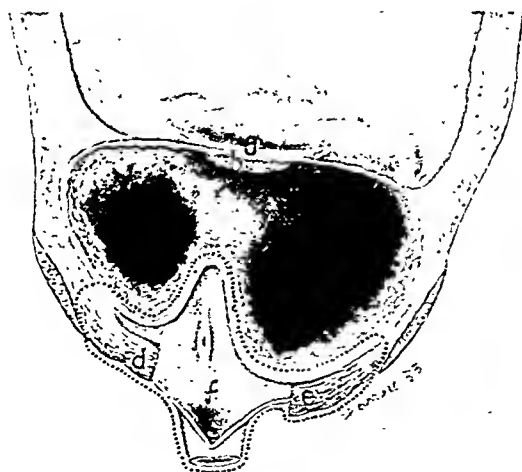


FIG. 299.—Same specimen as Fig. 298 after enucleation of the prostate. The heavy white line outlines the resulting cavity. It will be noted that the membranous urethra, the verumontanum, and the prostatic urethra around and distal to it have been left intact. The anterior commissure has been divided in this specimen for the purpose of exposing the interior of the prostatic urethra; during an actual operation it is left intact. The dotted black line surrounds the additional area which is commonly removed when the Freyer, particularly the open instrumental, method of prostatectomy is employed. *a*, The inter-ureteric bar; *b*, The trigonal muscle; *c*, The membranous urethra; *d* and *e*, The cut edges of the muscle of the anterior commissure; *f*, The verumontanum. (Semi-diagrammatic.)

can be cleanly and speedily removed with the greatest gentleness and facility. This result is accomplished with an almost complete absence of bruising and with a minimum of disturbance of the surrounding tissues.

FIG. 300.—Drawing of post-mortem specimen of bladder and prostatic urethra opened from the front, and showing lateral lobe enlargement of the prostate with a perfectly normal posterior commissure.

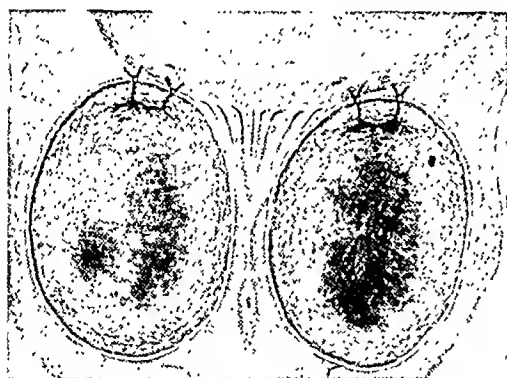
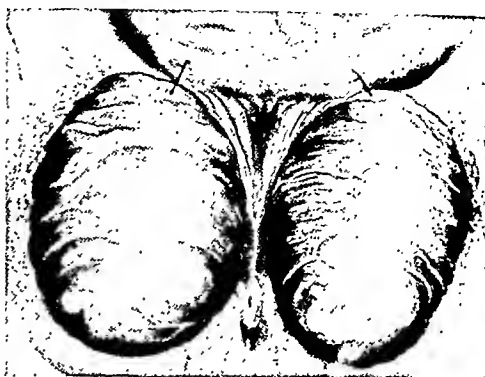


FIG. 301.—Same case as Fig. 300 after removal of the lateral lobes. The trigone has remained undisturbed in its normal position. Two haemostatic sutures are shown on each side. (Semi-diagrammatic.)

FIG. 302.—From the same specimen as Figs. 298, 299. The trigonal tongue is shown sutured in position as in the author's operation. (Pre-inserted here for ready comparison with the previous figure and to emphasize how closely reproduced is the normal anatomical relationship of the trigone and prostatic urethra.) (Semi-diagrammatic.)



6. Visualization of the prostatic cavity and removal of any adenomatous remnants or tags.

7. Insertion of the individual haemostatic sutures in the postero-lateral segment of the prostatic rim to control the gross haemorrhage (*see below*).

When, as occasionally happens, the lateral lobes are enlarged without any involvement whatever of the posterior commissure (*Fig. 300*), it will sometimes be found that the enucleation has resulted in the separate removal of each lateral lobe, the trigone being left intact and undisturbed (*Figs. 301, 302*).

8. Reconstruction of the floor of the prostatic urethra by suturing a wide muscular tongue of trigone into the prostatic cavity (*see below*). No. 3 plain catgut is used for these sutures. This step, so far from producing deformity as claimed by some critics, not only re-forms the floor of the prostatic urethra, but also straightens out the trigone, gives to its muscles a 'point d'appui', and puts it in position to resume its physiological rôle of pulling open the internal urethral orifice during the act of micturition (*Fig. 302*).

9. Obliteration of the prostatic cavity and re-formation of the side walls of the new prostatic urethra by two deep anterior transverse sutures of No. 3 catgut; these also finally control the venous oozing which normally occurs from the prostatic cavity after prostatectomy. Very rarely a third suture may be required when a very large cavity is present (*see below*). The newly formed prostatic urethra is always left open wide enough to admit the tip of the first finger, which is generally passed through it to guide the catheter up into the bladder during its insertion at the end of the plastic portion of the operation. All bleeding should be controlled before the catheter is passed. The catheter itself, as indicated above, plays no part in the control of hæmorrhage, ample room always being left round it to allow of drainage upwards into the bladder from the remnant of the prostatic cavity. Experience has proved that tight suturing of the prostatic rim round the catheter predisposes to sloughing of the contiguous parts and to secondary hæmorrhage.

10. Insertion through the urethra into the bladder of a rubber catheter, size 22 F., preparation of its vesical end, and transfixion by a needle armed with silkworm gut (*see below*).

11. Valvular closure of the bladder, obliteration of the space of Retzius, and closure of the lower angle of the incision in the aponeurosis by one single three-looped extended figure-of-eight suture of No. 3 plain catgut (*see Fig. 308*). This suture has been described in detail in previous publications. If, for any reason, an unduly large incision has been made in the bladder, the lower angle is first closed separately by one or, if necessary, more figure-of-eight sutures. These serve both to bring the muscle of the bladder wall together and to invert the cut edges in this situation. The remainder of the bladder incision is then closed by the single three-looped suture.

12. Complete closure of the abdominal wound.

13. Suspension of the vesical end of the catheter by tying the silkworm gut transfixion suture round a glass rod lying on the abdomen (*see Fig. 308*).

The Postero-lateral Hæmostatic Sutures.—From two to four sutures of No. 2 plain catgut are placed on either side of the mid-line in the posterior third of the rim of the prostatic cavity (*see Figs. 301, 302, 304*), which, in a properly conducted prostatectomy, is the site of the gross hæmorrhage in the vast majority of cases. Where two sutures have been placed on one side and gross bleeding is still taking place from the region between these

two sutures, the loose ends of these sutures may be tied together. Great care should be taken when two sutures are tied together in this way that no undue narrowing of the prostatic rim takes place in the direction of its circumference, as this would make it more difficult to draw down the trigonal tongue, and would thus predispose to ledge formation. Should bleeding still persist in front of or behind these sutures, further sutures should be placed in front of or behind those already placed. In no circumstances is a postero-lateral suture of one side tied across the mid-line to a postero-lateral suture on the opposite side, as this would deliberately encourage ledge formation.

After bleeding has been controlled in the region served by the postero-lateral sutures, gross bleeding may occasionally be seen from a large artery or vein lying deeply on the more anterior portion of the prostatic rim. When this occurs an individual suture should be placed around the bleeding point with the boomerang needle, since the anterior oblitative sutures when they are tied may not afford sufficient compression. All bleeding from the prostatic rim must be definitely controlled before proceeding further.

As the author has pointed out in previous papers, when the bleeding is arterial, the sutures should be placed in front of the bleeding point; when venous, behind.

The Posterior or Trigonal Suture.—In all cases the needle for the trigonal suture now embraces a much wider extent of tissue than described in previous communications. The point of entry of the needle is well down behind the

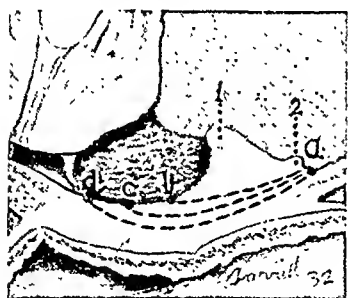


FIG. 303.—Sectional view showing needle tracks, ab, ac, ad, for three satisfactory grades of trigonal flap. 1, Torn apex of trigone. 2, Inter-ureteric bar; ab and sometimes ac do not require the use of the special capsule forceps; for ad, which yields the best results, the capsule forceps are essential.

base of the trigone (*see Fig. 304*). It is, of course, necessary to visualize the ureteral orifices before inserting the needle. If this is done, no fear need be entertained of compression of them when the suture is tied. The point of the needle is made to emerge in the prostatic cavity as far forward, i.e., as deeply, as possible.

For record purposes the author grades the trigonal flap according to its depth of penetration into the prostatic cavity as 'A', 'B', or 'C'.

In 'C' the flap is pulled well down into the prostatic cavity, almost completely reforming the floor of the prostatic urethra (*see Fig. 302 and Fig. 303 ad*). In 'B' (*Fig. 303, ac*) the flap penetrates not so deeply, and in 'A' (*Fig. 303, ab*) the flap is sewn down even

more superficially, but at the least in such a way that the horizontal muscular shelf or projecting ledge, which appears in this position after removal of the prostate, is converted into a steep vertical gutter. This gutter will be even further accentuated when the anterior transverse sutures are tied (*see Fig. 307*) and the flap becomes firmly bedded in position (*see below*).

The employment of flap 'A' or 'B' makes for greater simplicity of the operative technique, since the boomerang needle may generally be passed deeply enough through the prostatic capsule to form one or other of these

flaps without the aid of the special capsule forceps which have been described in previous publications.

Except that these more superficial methods of fixing the trigonal flap carry a considerably greater liability to suprapubic leakage of urine after removal of the catheter on the tenth day, the ultimate results seem to be as good as when flap 'C' is made. Experience has shown that, *ceteris paribus*, the better the flap, the less is the leakage.

Although the author's preference is very decidedly for the employment of the capsule forceps and of flap 'C' (Fig. 304), where this is possible, he has

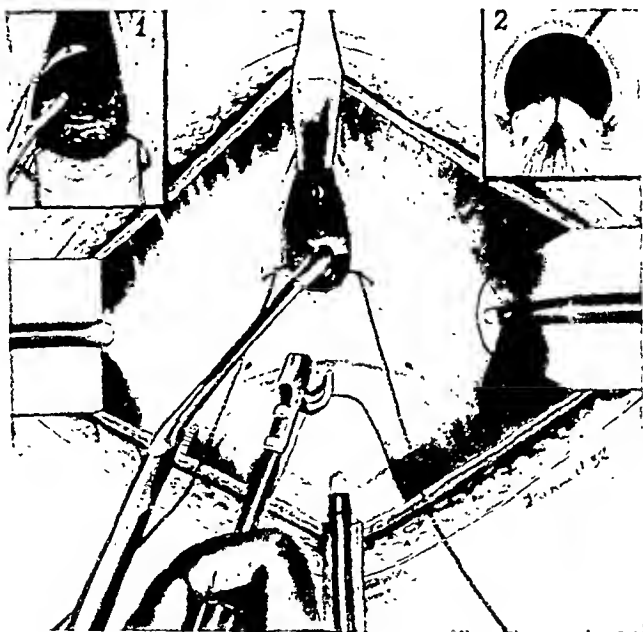


FIG. 304.—Formation of the trigonal flap: the needle enters behind the interureteric bar and emerges through the prostatic capsule at b. The special capsule forceps are picking up the prostatic capsule. Two hæmostatic sutures only are shown for the sake of simplicity. The new locking device for the boomerang needle is shown on the needle-holder.

Inset 1 shows the point of the needle emerging through the prostatic capsule: the suture carrier in position.

Inset 2 shows the trigonal suture tied, drawing down into the prostatic cavity the entire thickness of the trigonal tongue with its subjacent muscle and re-forming the floor of the prostatic urethra.

carried out a considerable number of operations employing flap 'A' or 'B' and has had no cause for regret.

So far as the risk of perforation of the rectum by the needle is concerned, this is a practical impossibility with the technique and needle described. This accident has never happened either in the author's hands, or, to the best of his knowledge, elsewhere.

During the tying of the trigonal suture the tip of the operator's left forefinger, if he is standing on the patient's left side (which is the author's practice)

should push down the knot as far as possible into the prostatic cavity (*Fig. 305*); this ensures that the trigonal tongue will be carried down to the lowest part of the loop when it is tied (*see Fig. 302*). If the right forefinger is used for this purpose, it will be found very difficult to pass this deeply enough into the prostatic cavity, and the tendency will be to draw the trigonal flap upwards away from the prostatic cavity when tying the knot.

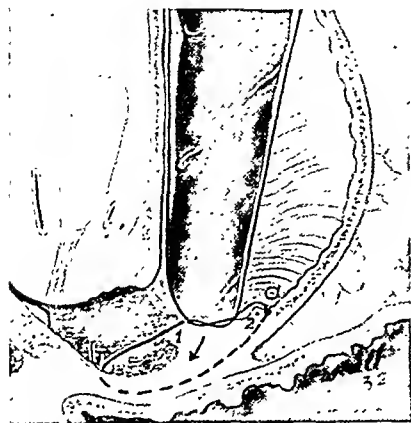


FIG. 305.—Sectional view showing the method of tying the trigonal suture, the left fore-finger pushing well down into the prostatic cavity. *ab*, Track of needle; 1, Torn apex of trigono; 2, Interureteric bar.

The Anterior Transverse Obliterative Sutures.—In the vast majority of cases two anterior transverse obliterative sutures are employed, though one may suffice for a very small cavity, and a particularly large cavity may very occasionally demand the use of three. One suture is tied before the next is placed.

The first, or most anterior suture, passes transversely at a tangent to the anterior segment of the prostatic rim as shown in *Fig. 306*. The second is parallel to the first and bisects what remains of the prostatic cavity. These sutures traverse the prostatic cavity deeply from side to side, just missing

its floor in the depths. They will lie above and in front of the catheter when it is passed.

Greater efficiency has been attained for these sutures by increasing the extent of their bite. The needle for each stitch now perforates the bladder base much more widely out on each side than formerly, and, in fact, takes about as wide a bite of the whole thickness of the bladder muscle in this situation as it can be made to hold. There results from this not only more nearly complete obliteration of the prostatic cavity, but also a wide inversion of the rim of the prostatic cavity and adjoining portion of the bladder base, thus largely reforming the side walls of the new prostatic urethra. During the process of tying these sutures the trigonal tongue sinks progressively to a lower plane, until finally it disappears almost entirely from view. It is thus made to penetrate even more deeply into its position in the prostatic cavity, in which it is now very firmly bedded (*Fig. 307*, inset). At the termination of the operation there should be no visible raw surface and no bleeding.

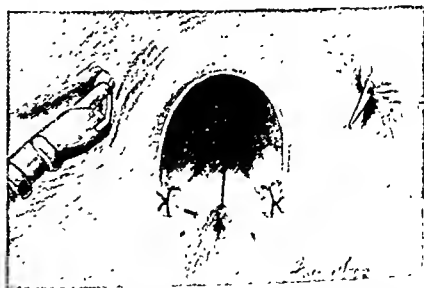


FIG. 306.—Needle in position for the first anterior transverse obliterative suture. Note the width of the bite of the needle. Trigonal tongue in position—two hamostatic sutures only are illustrated for the sake of simplicity.

Preparation of Retained Catheter.—A thin-walled rubber catheter of size 22 F. is employed. This is measured by gauge and not by the maker's marking.

After the catheter is passed into the bladder a second eye is cut about one inch from the tip, and the end of the catheter is cut off transversely just beyond this new eye. The full calibre of the catheter is thus made available for drainage (*Fig. 307*).

It has been found important to make sure that, owing to faulty manufacture, there is no narrowing at any point throughout the lumen of the

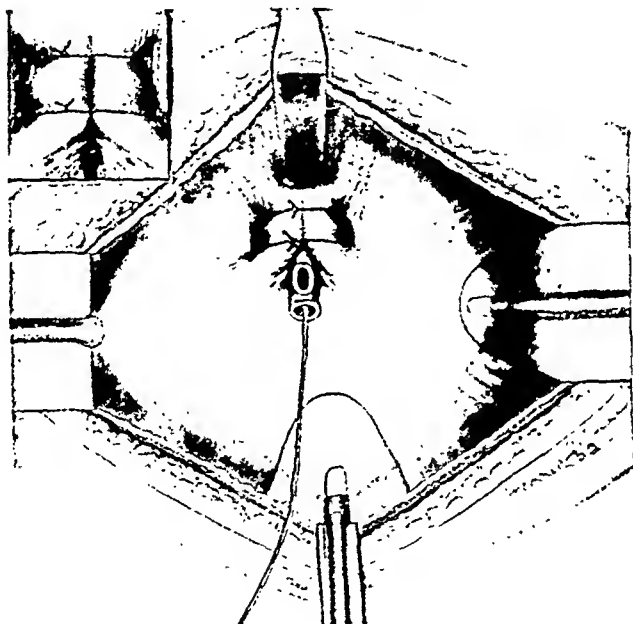


FIG. 307.—Second transverse obliterative suture inserted: plastic operation completed: catheter with silkworm-gut transfixion suture in position. The catheter is passed intact and the tip cut off after a second eye has been made.

Inset: Same stage before passage of the catheter. Note that there is no visible raw surface, that the trigonal flap lies on a plane below that of the rest of the bladder base and is firmly bedded in position, and that the lateral edges of the prostatic rim are deeply inverted, thus partly re-forming the side walls of the new prostatic urethra.

catheter. This was the cause of inefficient drainage in two of the author's cases. All catheters are now tested before use by passing through them a full-size obturator.

If, as occasionally happens, it is found that the catheter is too large to pass through the urethra into the bladder, no apprehension need be felt, as the catheter plays no part either in the control of hæmorrhage or in the urethral reconstruction. Suprapubic drainage should be instituted, and the bladder will be found to close rapidly after removal of the suprapubic drainage tube. As a matter of fact, the complete closure, though a valuable asset,

is rather in the nature of a comfort and convenience to the patient than a vital factor in the final outcome.

The operation is completed as shown in *Fig. 308*.

Prostatectomy in the Presence of Diverticulum of the Bladder.—When the diverticulum is of such a size that it can be dealt with from within the bladder, the complete operation (i.e., diverticulectomy plus prostatectomy) may be undertaken at the same sitting, provided it has been possible by preliminary preparation to cleanse the diverticulum efficiently of its foul content. When the diverticulum is larger and demands extravescical removal, or, in any case, when the content remains foul in spite of treatment, the

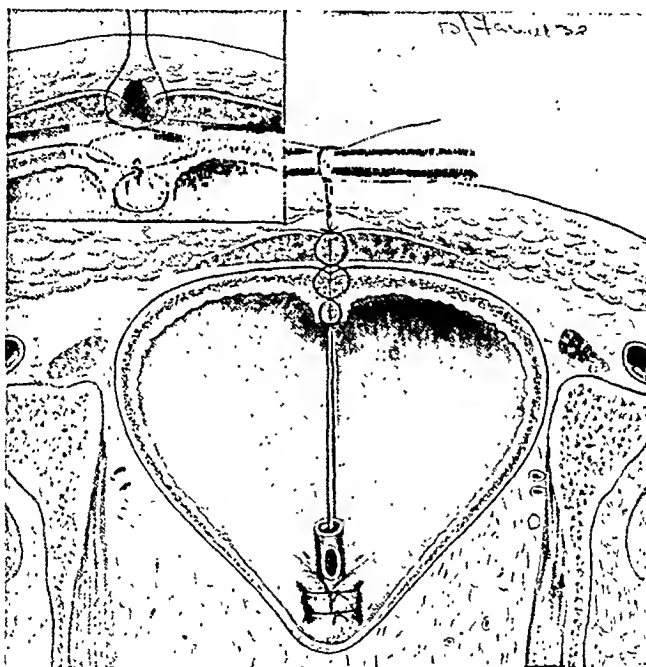


FIG. 308.—Sectional view of the completed operation seen from below. The three-loop suture is tied, approximating the bladder muscle, making a valvular inversion of the cut edges, obliterating the space of Retzius, and closing the lower angle of the incision in the aponeurosis. The catheter is tethered to the glass rod lying on the abdomen.

Inset: The three-loop or extended figure-of-eight suture, partially tightened. The catheter suture has been omitted to avoid confusion.

author prefers as a rule to remove the diverticulum at a preliminary operation in conjunction with cystostomy if the patient's condition warrants it, the prostatectomy being deferred to a later stage. In some cases, however, when the general condition of the patient was not such as to warrant any prolonged procedure, he has combined a plastic widening of the diverticular stoma, generally by means of a vertical downward incision terminating as close as possible to the internal urethral orifice, with prostatectomy and the usual plastic procedure on the prostatic cavity. The advisability of associated suprapubic drainage in cases of this type depends on the foulness or otherwise of the diverticular content.

Paraffin Method of Cleansing a Foul Diverticulum.—A diverticulum with offensive contents may often be rapidly and efficiently cleaned up by injecting a sufficient quantity of liquid paraffin into the bladder, after its routine daily irrigation, thoroughly to distend the diverticulum. When the excess is allowed to flow out of the bladder, the diverticular orifice will generally contract down and retain the remnant of oil in place of its previous content of decomposing urine.

Two-stage Prostatectomy with Closure.—A preliminary cystostomy is practised only when either catheterization is impracticable or it has been impossible to bring about sufficient improvement by catheterization to warrant the one-stage operation.

The prostatectomy is not undertaken until at least one month after the cystostomy in order to allow of subsidence of the wound induration. The patient in the interval is up and about with a small-gauge permanent cystostomy tube in position. When the prostatectomy is undertaken a vertical incision is carried downwards from the cystostomy opening, which, in these cases, has been made through a transverse skin incision two inches above the level of the symphysis. The same plastic technique on the prostatic cavity is carried out as in the one-stage operation.

A second set of bladder retractors, of the same design though half the width of those described above, has been made for use in the occasional case in which the latter cannot be introduced through the more restricted space available in the two-stage operation.

Water-tight closure of the abdominal wall is, of course, not so readily obtained in these cases. It is surprising, however, how little urinary leakage actually occurs. The leakage in this series of cases persisted in few beyond the twelfth and in none beyond the twenty-eight day.

The Post-operative Catheter Drainage.—At the termination of the operation, and before the patient leaves the operating table, a few syringe-fuls of 1-5000 oxycyanide of mercury solution are washed backwards and forwards through the catheter to remove the blood-clot which invariably will be found to have formed in the catheter. A wide-bore glass connection is then placed in the funnel end of the catheter and inserted into a sterilized 8-oz. glass bottle, into which some of the oxycyanide of mercury solution flows back from the bladder. When the patient is returned to bed the catheter is connected up in the usual way by a length of rubber tubing to a bottle at the bedside.

A careful watch must be kept on the catheter, especially for the first twenty-four or thirty-six hours, to ensure continuity of drainage. When the operation has been properly carried out, with all due attention to hæmostasis, there will rarely be any cause for anxiety in this regard. It is axiomatic that no bladder should ever be completely closed when the bleeding has not been adequately controlled. On this point there should be no room for errors of judgement.

The majority of patients require no interference with the catheter, but should any doubt be entertained about the continuity of the drainage there should be no hesitation in injecting, repeatedly if necessary, $\frac{1}{2}$ oz. of 1-3000 solution of silver nitrate to free the catheter of possible clots.

Irrigation of the bladder, beyond the amount necessary to free the catheter of clot, is neither necessary nor permitted.

Spasmodic and painful contractions of the bladder sometimes occur during the first two or three days of convalescence, especially in fat patients. This can generally be overcome or minimized by leading the rubber drainage tube below instead of over the patient's thighs.

The catheter is retained in position until the tenth post-operative day. It is removed by cutting across the silkworm-gut suture immediately below the glass rod. The hairpin-shaped remnant of silkworm gut comes away with the catheter. Most patients are out of bed on the eleventh day.

Post-operative Hæmorrhage.—Post-operative hæmorrhage, reactionary or secondary, of sufficient severity to demand cystostomy has been extremely rare throughout the series. It has occurred only once in the last 273 cases.

An injection into the buttock of from 1 to 2 oz. of whole blood, which need not be typed for this purpose, has seemed of value in the few cases in which its use was called for on account of the persistent formation of clots interfering with the continuity of drainage through the catheter, either before or after prostatectomy.

SUMMARY.

1. The author's operation of suprapubic prostatectomy with closure is described in detail.

2. The results obtained during the five years' currency of this operation down to October, 1932, are presented.

3. The number of cases submitted to prostatectomy was 371, the deaths were 10, the mortality rate was 2·7 per cent.

4. The operation is decidedly not one for the occasional prostatectomist, but the technique will present no insuperable difficulties to those experienced in this branch of surgery.

5. The essential features of the operation are the immediate control of hæmorrhage by suture, the re-formation of the prostatic urethra, and obliteration of the prostatic cavity, combined with immediate closure of the bladder and abdominal wound.

6. In addition to the improvement of mortality, morbidity, and operability rates, manifested by this operation as compared with other widely practised methods of suprapubic prostatectomy, this operation also completely obviates post-operative recurrence of obstruction. Further, the various septic manifestations, which have been the bugbear of prostatectomy, have almost completely disappeared.

REFERENCES.

¹ *Med. Jour. of Australia*, 1927, March 26. 460.

² *Surg. Gynecol. and Obst.*, 1930, Jan., 251.

³ *Med. Jour. of Australia*, 1927, Oct.

⁴ *Brit. Jour. Urol.*, 1929, Sept., 285.

⁵ *Brit. Med. Jour.*, 1932, ii, 192.

ENLARGEMENT OF THE ANTERIOR PORTION OF THE PROSTATE GLAND.

By BRENNAN S. CRAN.

CARNEGIE TEACHING FELLOW, DEPARTMENT OF PATHOLOGY, UNIVERSITY OF ABERDEEN.

THE following case of enlarged prostate is of interest in view of the unusual involvement of the anterior portion. Very few similar cases have been recorded (Wade,¹ Young,² and Walker³). In a series of 77 prostate glands removed at autopsy in which simple enlargement was present I found involvement of the anterior portion in only one, and Riches and Muir¹ in their series (59 cases) report an incidence of 1 per cent.

The prostate gland is composed of five lobes: two symmetrical lateral lobes, an anterior lobe in front of the urethra, a posterior lobe behind and below the ejaculatory ducts, and a middle lobe situated in the acute angle between the posterior urethral wall and the ejaculatory ducts. This classification has an embryological basis, notwithstanding the fact that before birth the demarcation between the lobes has disappeared with the exception of the posterior mid-prostatic groove which separates the lateral lobes. As is well known, simple enlargement affects the median or lateral lobes, and, in my experience, practically always both, although enlargement of one of them may predominate.

CASE REPORT.

Unfortunately no history is available, as the patient, aged 71, was admitted to the Aberdeen Royal Infirmary in a comatose condition, with laceration of the brain and fracture of the skull resulting from an accident; he died without regaining consciousness.

POST-MORTEM FINDINGS.—At the autopsy, on opening the bladder, which was moderately distended with urine, an enlarged prostate was found with an intravesical projection of about the size of a fairly large marble; this overhanging and partly occluded the internal meatus which lay behind it. The median and lateral lobes, which were but very slightly enlarged, did not produce an elevation of the floor of the bladder, the walls of which showed a moderate degree of muscular hypertrophy with trabeculation. A median sagittal section (*Fig. 309*) through the prostate demonstrated that the projection was not of polypoid nature, but part of a greatly enlarged anterior lobe which as a whole was approximately of the size of a walnut. The cut surface was studded with well-formed spheroids composed of hyperplastic prostatic glandular tissue, and, in all respects, its appearance was identical with that of a median- or lateral-lobe enlargement. The prostatic urethra was displaced backwards by the mass and formed a gentle curve. The posterior lobe was a trifle large for the age of the patient. Microscopic examination of the anterior lobe showed changes exactly analogous to those met with in the typical median- and lateral-lobe enlargement. The ureters, pelvis, and kidneys presented no unusual changes, except deep passive congestion of the last named. There was no cystitis. No stricture of the urethra was present.

Wade's and Young's cases are illustrated by a photograph and drawing respectively, but, though admitting that this type of enlargement is rare, these writers give no further detailed account of these cases. In Wade's specimen "the disease was confined to the anterior lobe", but in Young's case the median lobe was also enlarged but to a less degree.

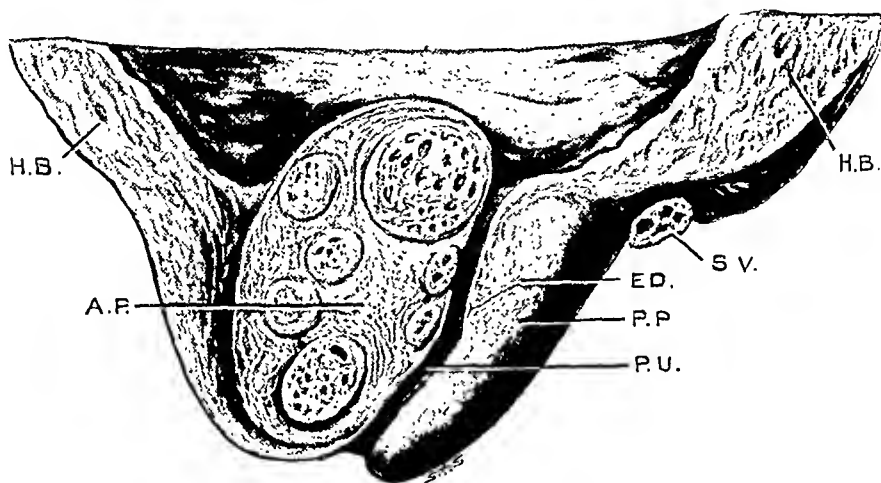


FIG. 309.—Median section showing the enlargement of the anterior portion of the prostate gland and the accompanying hypertrophy of the bladder. H.B., Hypertrophied bladder wall; A.P., Enlarged anterior portion of the prostate gland showing the intra-vesical projection and the well-formed cystic 'spheroids'; P.U., Prostatic urethra; P.P., Posterior portion of the prostate gland; S.V., Portion of a seminal vesicle; E.D., Terminal portion of an ejaculatory duct. ($\times 2$.)

COMMENTARY.

1. Developmentally the glandules of the anterior lobe of the prostate are the first to cease growth and retrogression is apparent in the earliest years of life. In view of this it is difficult to understand why in exceptional cases these structures persist to an unusual extent and in later life may even become the site of hyperplastic changes, especially as no glandular deficiency is apparent in the rest of the organ, when such exuberance might be regarded as of a compensatory nature.

2. Anterior-lobe enlargement in itself is capable of causing urinary obstruction. This is well shown in my case by the presence of the hypertrophy of the bladder, no other likely causal lesion being found at the autopsy. This would tend to prove that interference with the integrity of the trigonal muscle is not a *sine qua non* in the causation of urinary obstruction in prostatic enlargement. During micturition the posterior segment of the bladder neck is drawn backwards so that the angle formed by the floor of the prostatic urethra and the trigone is increased, the one gradually meeting the other instead of at an acute angle; the anterior segment remains stationary. This mechanism is due to the contraction of the muscular fibres of the trigone, which run from their origin near the inter-ureteric bar to be inserted in the verumontanum.

It has recently been suggested by Walker⁵ and others that an important cause of urinary obstruction in enlargement is interference with the action of this muscle, whether by infiltration of its fibres or by an increase in the size of the angle between the trigone and the floor of the urethra. Whilst this view undoubtedly must be true in many cases, especially in median-lobe hyperplasias and cases included under the term of 'prostatic bar', it cannot be universally so, as in the case I have described the disease was practically confined to the anterior segment of the bladder neck, and the trigone and floor of the posterior urethra were apparently unaffected.

It is probable that frequency of micturition had been present during life owing to the presence of the intravesical projection formed by the large anterior lobe, which, apart from playing the rôle of a foreign body within the bladder, undoubtedly would have caused stretching and irritation of the mucous membrane of the bladder neck. The mucosa in front of the meatus is, however, less sensitive than that covering the trigone, which is the part affected in median-lobe enlargement.

3. Rectal examination would fail to demonstrate the presence of an enlarged anterior lobe, although this would readily be seen on cystoscopy.

I wish to thank Professor Shennan for permission to publish this case.

REFERENCES.

- ¹ WADE, H., *Ann. of Surg.*, 1914, lix, 321.
- ² YOUNG, H. H., *Practice of Urology*, 1926, i, 424.
- ³ WALKER, K., *Diseases of the Male Organs of Generation*, 1923.
- ⁴ RICHES, E. W., and MUIR, E. G., *Brit. Jour. Surg.*, 1933, xx, 366.
- ⁵ WALKER, K., *Brit. Med. Jour.*, 1933, i, 355.

AN INQUIRY INTO THE RESULTS OF SURGICAL TREATMENT OF GENITAL TUBERCULOSIS IN THE MALE.

By RALPH O. LEE,

RESIDENT ASSISTANT SURGEON, ST. THOMAS'S HOSPITAL;

AND KENNETH BOWES,

SURGICAL REGISTRAR, ST. THOMAS'S HOSPITAL.

GENITAL tuberculosis is a local manifestation of a general disease the ultimate cure of which depends upon the resistance of the patient to the attacking organism. General treatment in the form of improved hygienic conditions and rest, coupled with the administration of tuberculin, has been shown in a minority of cases to produce cure, but in the majority it fails to stop the spread of the disease through the seminal tract. Local rest without surgical interference cannot be obtained, and there can be little doubt that surgery used to remove obviously diseased organs is completely justified.

Published results of series of cases treated without operation are scanty but uniformly depressing. Barney¹ quotes 11 cases, 6 of which died of tuberculosis. Keyes² gives 30 per cent as his figure for cure in 24 cases, and Lelongt³ using tuberculin claimed 33 per cent cures in 70 cases.

With surgical interference there is a distinct improvement. Orchidectomy and, more recently, epididymectomy have been the operations most widely practised, and numerous series of results have been published. These are difficult to compare, since the standard accepted for cure, and the length of time for which cases have been followed, vary with different authors. Simon,⁴ in 107 cases treated by orchidectomy, claimed 57 per cent cured, no period of observation being given. Lapeyre,⁵ who watched cases four to ten years after operation, claimed 75 per cent cures. Bruns,⁶ reporting 111 cases, gave 40 per cent as cured in three to thirty-four years after operation, and out of 78 unilateral cases 60 per cent had the opposite side affected within four months of operation. Boguljuboff,⁷ in 166 unilateral cases, showed a recurrence rate of 76 per cent, and Berger⁸ had a corresponding rate of 60·4 per cent. Barney¹ quotes 49 cases of unilateral disease. Within three years of orchidectomy 37 per cent had the opposite side affected. Keyes,² in a similar series, gave a figure of 60·9 per cent. Young⁹ gives a series of 85 cases treated by simple orchidectomy which have been thoroughly studied, and a summary of his findings is given below:—

	Per cent
Remained well or improved	46
Recurrence in opposite epididymis	31*
Recurrence in prostate or seminal vesicles	3·4
Remote mortality	29
Operative mortality	Nil

* Of 45 unilateral cases.

The tendency to recurrence after orchidectomy, already well-known, is strikingly illustrated by these figures. This, and the remote mortality, has led surgeons to consider more radical procedures. Young is of the opinion that the disease begins centrally in the prostate and seminal vesicles, and strongly advocates a wide excision including the epididymis, vas deferens, one or both seminal vesicles, and portions of the prostate if these latter are involved.

If thoroughly carried out this is an operation of considerable magnitude, and in practice it is rarely feasible to do more than remove the diseased vesicle with the vas and epididymis in one piece. The problem therefore is: Does the removal of a diseased vesicle materially improve the prognosis? Young has published 24 cases treated by his radical operation. His figures show:—

	Per cent
Alive and well (up to 9 years after operation) ..	54
Recurrence in opposite epididymis (7 unilateral cases) ..	42
Remote mortality	20
Operative mortality	4

Of other observers who have published figures of the radical operation, Baudet¹⁰ in 58 cases claimed 78 per cent cured, one to six years after operation. Quinby¹¹ states that he performed the operation in 7 cases with good immediate results, but gives no after history. Whiteside,¹² however, quotes 22 cases, none of which were watched for more than two years, in which he considers the results are poor.

In order to determine the significance of vesicular enlargement and the value of this more radical treatment, a series of cases taken from a ten-year period has been followed and analysed. The details of most of the cases have been obtained from the records of St. Thomas's Hospital and the remainder are due to the kindness of Mr. C. A. R. Nitch. Eighty-nine cases which came under treatment during the years 1921–30 inclusive have been traced during 1932. Most of these were seen and examined by one of us, and wherever possible the urine was examined for the presence of pus and tubercle bacilli. The majority were subjected to limited operations, either orchidectomy or epididymectomy, but a smaller group underwent radical orchido-vaso-vesiculectomy. At the same time the opportunity has been taken to investigate the mortality of the disease, and the liability to ascending infection of the urinary tract.

The clinical extent of the disease when the patients first came under observation is given in *Tables I* and *II*. *Table I* contains primary genital cases, and *Table II* cases secondary to renal tuberculosis. Of the total 89, 48 showed enlargement of one or both seminal vesicles.

Table I.—PRIMARY GENITAL CASES.

Unilateral epididymitis alone	32
Unilateral epididymitis with involvement of prostate or vesicles ..	40
Bilateral epididymitis alone	6
Bilateral epididymitis with involvement of prostate or vesicles ..	2
Total	80

Table II.—CASES SECONDARY TO RENAL TUBERCULOSIS.

Unilateral epididymitis alone	2
Unilateral epididymitis with involvement of prostate or vesicles	5
Bilateral epididymitis alone	1
Bilateral epididymitis with involvement of prostate or vesicles	1
Total				9

Mortality.—The true mortality of the disease in the series was difficult to estimate, since, in cases dying some years after treatment, the cause of death could not always be ascertained. An analysis of the fatal cases is given in *Table III*.

Table III.—ANALYSIS OF FATAL CASES.

<i>Immediate</i>	Post operation	1
	Pulmonary tuberculosis	7
<i>Remote</i>	Miliary tuberculosis	1
	Renal tuberculosis	5
	Probable tuberculosis	5
	Unascertained	5
					19

The patients included in 'probable tuberculosis' were known to have active disease when last heard from. The mortality from all causes during the period two to twelve years of observation is 21.3 per cent. The mortality from known or probable tuberculosis is 17 per cent. The only post-operative death followed a radical operation and was due to tuberculous bronchopneumonia.

Ascending Infection of the Urinary Tract.—The close connection between genital and renal tuberculosis has long been recognized, with spread from kidney to epididymis as the usual route. It is a striking fact in this series that only 9 of 89 cases followed renal disease. The incidence of the reverse spread from genital to urinary tract is smaller still. In order to estimate this the urine of 50 patients was examined microscopically for the presence of pus and tubercle bacilli, specimens from twenty-four hour collections being used. In 40 cases the urine was clear. In 8 cases pus without tubercle bacilli was found, and in all of these there were no symptoms, the patients being otherwise well. In only 2 cases was pus with tubercle bacilli found. One of these cases followed renal tuberculosis; the other had had both testes removed for tubercle, the first in 1920; and in May, 1932, urinary investigation revealed a tuberculous pyonephrosis with much calcification. In view of the long previous involvement of the first testis, it is possible that the renal condition was secondary. This is the only example in 50 cases in which ascending infection of the urinary tract can be alleged.

Treatment.—The cases have been divided into four groups, and a classification modelled on that of Young has been used.

1. In the first group are 11 cases which did not undergo operation; in the majority this was because of the presence of active pulmonary disease. The diagnosis in the remainder was established on clinical grounds only, and may therefore be open to doubt. The present condition of this group of cases is given in *Table IV*.

Table IV.—CONSERVATIVE TREATMENT.

Apparently inactive	6
Apparently active	1 (bilateral)
Died of phthisis	4
Total				11

In most of these cases tuberculin was not used, and no estimate of its value can be given. It is noteworthy that over half of the cases are apparently inactive two to twelve years after first observation.

2. The second group contains 62 cases which were treated by a limited operation—either orchidectomy or epididymectomy; 21 of these cases showed vesicular enlargement prior to operation. The present condition of the group is given in *Table V*.

Table V.—LIMITED OPERATION (62 CASES).

Apparently cured	29 (46.6 per cent) (6 bilateral)
Recurrence in prostate or vesicles	3 (4.9 per cent) (1 bilateral)
Recurrence in opposite epididymis	22 (35.5 per cent)
Died without further urogenital lesions	8 (13.0 per cent) (1 bilateral)

Gross recurrence rate, 49.3 per cent.

Recurrence rate in opposite epididymis in 54 unilateral cases, 40.7 per cent.

3. The third group contains the 21 of these cases in which vesicular enlargement was noted. *Table VI* shows an analysis for comparison.

Table VI.—LIMITED OPERATION IN 21 CASES IN WHICH
VESICULAR ENLARGEMENT WAS NOTED.

Apparently cured	10 (47.6 per cent) (2 bilateral)
Recurrence in opposite epididymis	8 (38.1 per cent)
Died without further genital lesions	3 (14.3 per cent)

Gross recurrence rate, 38.1 per cent.

Recurrence rate in opposite epididymis in 19 unilateral cases, 42.1 per cent.

It is surprising that both liability to spread to the contralateral epididymis and gross recurrence rate are no higher than in the main group. In other words, enlargement of the seminal vesicles appears not to affect the prognosis.

4. The fourth group is composed of 16 cases in which radical operations were performed. Vesicular enlargement was present in all, and the vesicles on removal were caseous and microscopically showed active tuberculosis. The operation in each case consisted in removal of the epididymis (or testis) with the vas and seminal vesicle, usually in one stage. Portions of the prostate were not removed. The present condition of this group is given in *Table VII*.

Table VII.—RADICAL OPERATION (16 CASES).

Apparently cured	9 (56.3 per cent) (1 bilateral)
Recurrence in opposite epididymis	6 (37.5 per cent)
Post-operative death	1 (6.2 per cent)

Gross recurrence rate in recovered cases, 40.1 per cent.

Recurrence rate in opposite epididymis in 14 unilateral cases which recovered, 42.9 per cent.

Fifty-six per cent of these patients are now well without further operation, a figure which compares favourably with any of the preceding series. The remote mortality has been nil, and although it is difficult to attribute this entirely to the operation, it is very satisfactory. On the other hand, there appears for the first time a post-operative mortality, and the figures for recurrence show no improvement compared with those of the more limited operation, but are indeed slightly worse.

SUMMARY.

1. A series of 89 cases of genital tuberculosis in the male, occurring in a ten-year period, has been followed up and analysed.
2. The mortality of the disease appears to be about 20 per cent.
3. Upward extension of infection to the urinary tract is rare.
4. The recurrence rate after surgical interference is about 40 per cent. This figure is not influenced by the presence of a palpable vesicle before operation, nor by its surgical removal.
5. Radical operation with vesiculectomy, whilst not affecting the recurrence rate in the other epididymis, seems to decrease the remote mortality.
6. In this series radical operation has introduced an immediate mortality.

We should like to express our thanks to the surgeons of St. Thomas's Hospital who have kindly allowed us to investigate their cases, and in particular to Mr. C. A. R. Nitch, who first suggested this research and without whose direction and valuable criticism it would not have been possible.

REFERENCES.

- ¹ BARNEY, "Genital Tuberculosis" in Cabot's *Modern Urology*, 1924, 532.
- ² KEYES, *Ann. of Surg.* 1907, June, 918.
- ³ LELONGT, *Thèse de Paris*, 1911, quoted by Barney.
- ⁴ SIMON, *Deut. Gesellschaft f. Chir.* 1901, xxx.
- ⁵ LAPEYRE, *Arch. gén. de Chir.*, 1912, viii.
- ⁶ BRUNS, *Deut. Gesellschaft f. Chir.*, 1901, xxx.
- ⁷ BOGULJUBOFF, quoted by Young, *Arch. of Surg.*, 1922, iv, 334.
- ⁸ BERGER, *Arch. f. klin. Chir.* 1901, lxxviii, 915.
- ⁹ YOUNG, *Practice of Urology*, 1926, i, 278; *Arch. of Surg.*, 1922, iv, 334; *Surg. Gynecol. and Obst.*, 1918, April, 375.
- ¹⁰ BAUDET, *Rev. de Chir.*, 1901, i.
- ¹¹ QUINBY, *Jour. Amer. Med. Assoc.*, 1918, Nov., 1790.
- ¹² WHITESIDE, *Calif. Jour. of Med.*, 1910 viii, 88; *Trans. Amer. Assoc. Genito-urin. Surg.*, 1914, ix; *North-western Med.*, 1919, xviii, 83.

CALCIFICATION, DECALCIFICATION, AND OSSIFICATION.***By R. WATSON JONES,****HON. ASSISTANT ORTHOPEDIC SURGEON, LIVERPOOL ROYAL INFIRMARY ;
LECTURER IN ORTHOPEDIC PATHOLOGY, LIVERPOOL UNIVERSITY ;****AND R. E. ROBERTS,****HON. RADIOLOGIST, LIVERPOOL ROYAL INFIRMARY ;
LECTURER IN RADIOLOGY, LIVERPOOL UNIVERSITY.****I. THE PATHOLOGY OF CALCIFICATION AND OSSIFICATION.**

THE problems of bone growth have for many years attracted the attention of physiologists, anatomists, and biochemists, and many controversies have centred on the nature of the osteoblast and the osteoclast, the function of the periosteum, the chemical constitution of the bone salt, and the factors determining the deposition of this salt in the skeletal framework. To the clinician and the surgeon these problems have appeared to be chiefly of academic interest, and in no other branch of medicine has there been more confused thought, more inaccurate observation, and more unsound teaching, than in the clinical pathology of ossification, calcification, and decalcification of tissues. Bone formation has been attributed to the specific activity of osteoblasts, despite the fact that bone may develop in tissues far distant from any possible source of osteoblasts. The deposition of calcium in abnormal situations has not been correlated with the deposition of calcium in normal bone, and special theories have been formulated to account for it.¹ No clinical distinction has been clearly established between destruction of bone and simple decalcification. The association of decalcification of bone with the hyperæmia of infection has been accepted, but the same decalcification arising after fracture of the neck of the femur and leading to non-union has been attributed to impairment of the blood-supply. An identical pathological entity has therefore been attributed to exactly opposite pathological processes.

Although experimental observation is still incomplete, the work which is already established is sufficient to enunciate principles of the most vital clinical significance. The osteoblast can no longer be regarded as a cell endowed with the specific power of laying down bone. It is merely the cell of a mesenchymatous tissue in which inorganic salts may be deposited, absorbed, redeposited, and reabsorbed according to the local influence of enzymes, and under the general control of endocrines. This is not a property

* Based on a paper by R. Watson Jones read before the Innominate Club, Liverpool, September, 1931, and a joint paper by R. Watson Jones and R. E. Roberts read before the Radiological Section of the Royal Society of Medicine, March, 1933.

peculiar to bone cells. A similar power to effect the deposition of inorganic salts is possessed at certain stages of development by the cells of cartilage and of fibrous tissue. The bone cell has no more specific power than the cartilage cell or the fibrous-tissue cell, and in fact all of these cells are of the same type. The early undifferentiated mesenchyme of the embryo forms a common mesoblastic stem from which may develop either fibrous tissue, cartilage, or bone, and the cells of these tissues are strictly comparable. The



FIG. 310.—Tuberculous disease of the os calcis with calcification of caseous abscess. Sinuses formed, secondary infection supervened, and areas of ossification developed. When the amorphous calcium drained away, the bone remained as sequestra and the sinuses persisted until they were removed. (*North Wales Sanatorium case.*)

bone cell of Purkinje is a fibroblast imprisoned in bone and in a state of quiescence. The osteoblast is a fibroblast enlarged because it is active. Any of these tissues, after preliminary de-differentiation to the state of primitive mesenchyme, may be built up again in the form of one of the other tissues. When the endocrine balance is disturbed as in hyperparathyroidism skeletal bone may be de-differentiated only to reappear as fibrous tissue. On the other hand, in myositis ossificans progressiva the fibrous tissue of tendons

and muscles is de-differentiated and built up again as bone. The change from cartilage to bone is seen in every epiphysial disc, and under pathological conditions the same process may be observed in the semilunar cartilages of the knee-joint (*see Fig. 340*). The view put forward by Leriche and Policard,⁸ and advocated in this country by Greig,¹⁰ is that the only factor necessary to determine bone as the ultimate destiny of a mass of primitive mesenchyme is an excess of calcium with an adequate blood-supply. By these factors the theory accounts for the development of bone not only in the skeleton but in abdominal scars, thoracic viscera, and other tissues. Any adult tissue which can be de-differentiated into primitive mesenchyme may be built up again as bone if there is calcium excess. Pathological calcareous deposits in any region of the body may therefore change into pathological ossific deposits if the blood-supply should become adequate (*Fig. 310*).

The Physico-chemical Mechanism of Calcification.—The mechanism of the deposition of calcium in these mesenchymatous tissues is obviously a physico-chemical problem. It is probable that the calcium is not separately precipitated in the form of calcium carbonate, calcium phosphate, and other salts, but that the bone salt is a complex calcium carbonato-phosphate molecule [e.g., $\text{Ca}_3(\text{PO}_4)_2 \cdot \text{CaCO}_3$] holding in combination magnesium, sodium, potassium, chloride, fluoride, and hydroxyl groups.¹¹⁻¹⁷ In addition to inorganic phosphate, blood contains a phosphoric ester the calcium salt of which is soluble. This ester cannot yield PO_4 ions on dissociation, so that it does not affect the saturation of the calcium phosphate.

Robison¹⁸ has shown that tissues which normally become the site of calcareous deposit contain an enzyme, phosphatase, which hydrolyses the ester and sets free inorganic phosphate. The increased concentration of PO_4 ions results in the precipitation of the carbonato-phosphate molecule. In a growing bone the epiphysial line has the greatest phosphatase activity, the already developed bone has a lower activity, and the resting epiphysial cartilage is completely inactive. The patella of a child before the appearance of the centre of ossification has no phosphatase, but so soon as ossification commences phosphatase is to be found. The phosphatase activity of tissues which become the site of pathological calcification has not yet been estimated except in the case of the kidney. But in cases of heterotopic ossification of the abdominal wall, experimentally induced in dogs by transplanting bladder epithelium, Huggins²² has shown that the ossifying tissue possesses very high phosphatase activity.

The activity of the enzyme is dependent on the hydrogen-ion concentration of the tissue fluids, and in the case of another phosphatase present in blood-corpuseles Martland²⁴ has shown that there is a delicate equilibrium. If the pH falls below 7.3, the esters are hydrolysed and the inorganic phosphates increase, whereas if the pH is above 7.35, the esters are synthesized and the inorganic phosphates decrease. Moreover, the hydrolysis of phosphoric esters will not necessarily be followed by calcification unless the concentration of calcium ions is also favourable. The kidney has a high phosphatase activity, but with blood calcium at the normal level of 10 mgrm. per cent no calcification is observed. In the presence of hypercalcaemia, calcification of the kidney is very frequently seen (*see Fig. 324*).

Pathological Calcification.—The deposition of calcium salts in mesenchymatous tissues would therefore appear to depend on the concentration of calcium ions and the activity of the phosphatases. The first factor is of significance in relation to the multiple calcareous deposits which may be associated with hypercalcaemia. This association was noted by Virchow and has been observed many times in recent experimental work with parathyroid extract.²⁵⁻³⁰ Blood may not be a supersaturated solution of the bone salt,



FIG. 311.—Calcification of caseous psoas abscess in a case of tuberculous disease of the spine. (*Shropshire Orthopaedic Hospital case.*)

but normally it is almost fully saturated. In hyperparathyroidism the serum concentration of calcium is increased by 50 per cent or more, and this would reasonably be expected to result in generalized precipitation in many tissues.

But supersaturation will not explain the ordinary case of pathological calcification where there is no evidence of hypercalcaemia. The factors governing phosphatase activity are not yet fully established, but there can

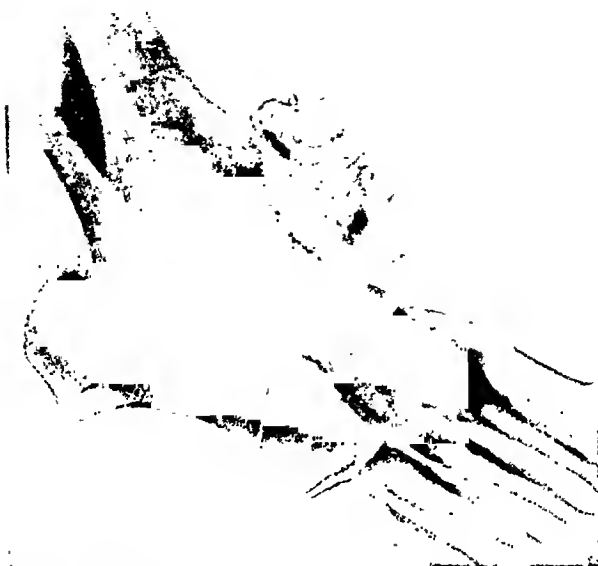


FIG. 312.—Calcification of fibroma of the tibialis anticus. (*Mr. C. Thurstan Holland.*)

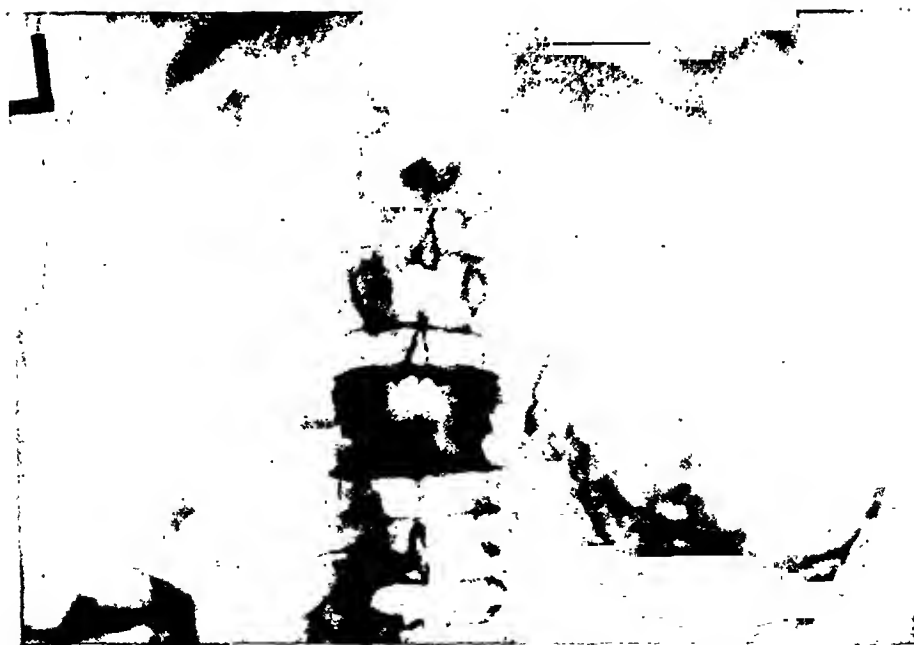


FIG. 313.—Calcification of hydatid cyst of liver distinguished from renal calcification by uroselectan.

be no doubt that the local circulation must play an important part, whether through its effect on the carbon dioxide content of the tissues, the hydrogen-ion concentration, or on other undetermined factors. Pathological calcification is seen in tissues of which the metabolism is normally low—in the avascular caseous masses of old tuberculous and syphilitic disease (*Fig. 311*), in tendons and ligaments,¹¹¹ in the falx cerebri and the fascia lata, in costal cartilages,³¹ the semilunar cartilages,¹⁴⁸ and the intervertebral discs,¹³³ and in the fibrous walls of blood-vessels. Even in such relatively avascular tissues the deposition of calcium is usually the result of still further reduction in the metabolic rate by fibrosis following trauma or infection. In the tendo Achillis calcification follows the fibrosis of subcutaneous tenotomy (p. 491), and in the supraspinatus tendon it follows chronic traumatic tendonitis (p. 484). In the pericardium calcification is seen after fibrous pericarditis, and in the falx cerebri after sinusitis. The calcification of tumours is observed when the blood-supply is so impaired that degenerative changes have appeared. It is therefore seen in the uterine fibroid, the fibroma of tendons⁴⁶ (*Fig. 312*), and in the fibrous walls of cysts (*Fig. 313*).

Whatever may be the association between impaired vascularity, phosphatase activity, and calcification, it is definitely reversible. Tissues may be calcified, decalcified, and recalcified by alternately decreasing the circulation through the influence of X rays and allowing it to increase by a cessation of treatment (p. 469). Moreover, pathological calcareous deposits resulting from reduced vascularity of the tissues, may reabsorb in the presence of secondary irritative hyperæmia (p. 485). If the deposit of calcium is sufficiently extensive, revascularization will result in ossification; but with smaller deposits, revascularization leads to complete reabsorption.

The Relation of Vascularity to Calcification of Bone.—There can be little doubt that the same factors govern the calcification of all mesenchymatous tissues, and the same relationship of vascularity to calcification which is observed in pathological deposits is also observed in bone. If the blood-supply to a bone is decreased, the bone undergoes increased calcification; if the blood-supply is increased, the bone undergoes decalcification.

Diminution of the arterial blood-supply of bone is constantly seen in syphilitic osteitis where there is endarteritis obliterans, and the characteristic bone change is one of increased calcification or sclerosis. Similarly increased calcification is observed in the later stages of osteomyelitis, when the initial hyperæmia has subsided and has been replaced by the ischæmia of reparative fibrosis.

The hyperæmia which causes decalcification may be infective in origin. it may be traumatic and due to the liberation of the vasodilators histamine and acetylcholine, or it may be of the nature of a relative hyperæmia from disuse. There appears at first sight to be some difficulty in correlating the decalcification of injury and infection, where obviously there is hyperæmia, with the decalcification of disuse. In the first place it must be recognized that disuse rarely stands alone, and as a rule there is the added factor either of injury or of infection. In a paralytic limb where there is uncomplicated disuse, decalcification is of relatively mild degree. Moreover, even in such a case, although there is total functional inactivity, there is relatively little

impairment of arterial supply, and a blood-supply which is normal for a limb in normal activity becomes a relative hyperæmia when the limb is totally inactive. The degree of decalcification is therefore proportionate to the intensity of the hyperæmia. It is least marked and develops most slowly from the relative hyperæmia of disuse, it is more severe with the greater hyperæmia of trauma, and it is most complete with the acute hyperæmia of infection.

A fact which is obvious, but which requires emphasis owing to the importance of its clinical applications, is that a fragment of bone completely cut off from the circulation so that it is avascular, will retain its original calcium content unchanged, whatever changes may occur in the adjacent vascular bone.

Résumé.—The pathological and biochemical observations which have been discussed and which are of clinical importance may be summarized as follows:—

1. There is normally a balance between the calcium content and the vascularity of mesenchymatous tissues (which is probably associated with phosphatase activity).

a. In the case of bone.—

Normal circulation	-----	Normal calcification
Increased blood-supply	---->	Decalcification
Decreased blood-supply	---->	Increased calcification
Blood-supply cut off	---->	Unchanged calcification

b. In any mesenchymatous tissue of low metabolism.—

Decreased blood-supply	---->	Pathological calcification.
<small>(Fibrosis of trauma or infection)</small>		

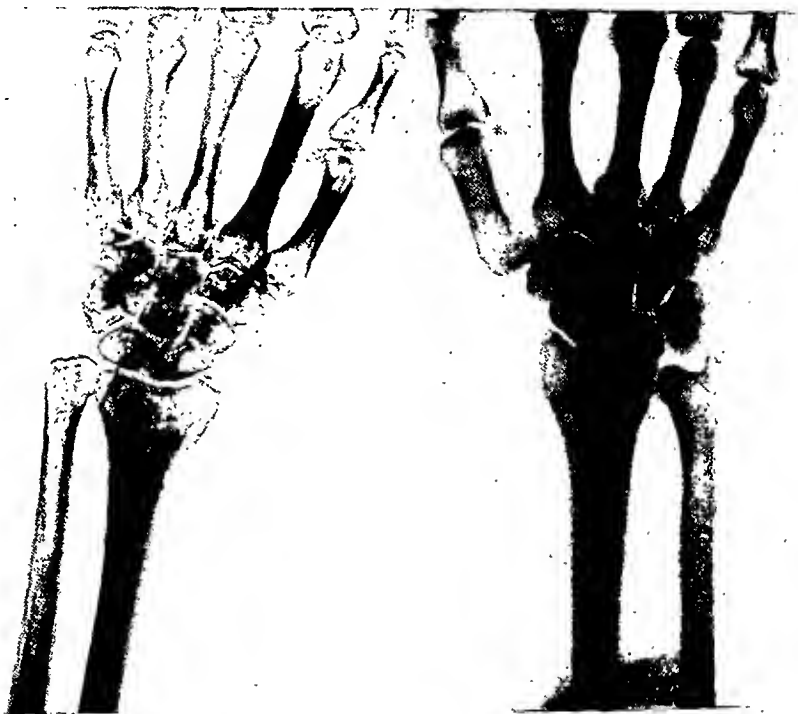
2. Fibroblasts + excess of calcium + adequate blood-supply ———> bone.

II. THE RADIOLOGICAL SIGNIFICANCE OF THE DENSITY OF BONE SHADOWS.

The pathologist recognizes that hyperæmia of bone produces decalcification, and anæmia produces sclerosis; the radiologist recognizes the converse, and must interpret decalcification as evidence of an increased blood-supply and sclerosis as evidence of a decreased blood-supply.

Disuse decalcification affects the whole of the bones of the limb equally, except that the vascular cancellous bone will obviously be more involved than the compact bone. The degree of decalcification may be taken as an accurate index of the functional use or disuse of the limb. *Fig. 314* shows two identical cases of Colles's fracture of the radius. The first has been immobilized by splints which have seriously interfered with the functional use of the limb. The second has been immobilized just as completely, but by means of a plaster cast which has left free the fingers and the palm of the hand. In the second case work has been continued throughout, so that there has been no diminution in muscular activity despite the immobilization of the joint. The first shows marked decalcification and the second shows none.

Very complete decalcification may be seen in the presence of infection of neighbouring parts, but it is important to recognize that the loss of calcium in such cases may be due entirely to hyperæmia, and does not necessarily imply any infection of the bone itself. In a recent case of tuberculous tenosynovitis of the tibialis posticus, there was such marked localized decalcification of the metaphysis and epiphysis of the tibia that the diagnosis of osteomyelitis seemed fully justified. But when the tuberculous sheath was excised the bone was found to be uninfected. Although it was decalcified and cut easily with a scalpel, there was neither inflammatory nor destructive change.



A

FIG. 314.—Disuse decalcification of bone. Two similar cases of Colles's fracture of the radius eight weeks after injury. A, In this case there has been excessive splintage and no functional activity. B, In this there has been full functional activity throughout.

In tuberculosis of the hip-joint, radiological examination in the active stage of the disease may suggest complete destruction of the femoral head. Nevertheless subsequent X-rays taken after quiescence of the infection and the resumption of function, prove that the bone had been merely decalcified. Cases which would otherwise appear to be remarkable examples of bone regeneration are not infrequently observed.

Similarly, in neoplasms of bone it is important to distinguish between destruction and simple decalcification. Bone sarcomata may be osteolytic or osteoplastic in nature. The relationship between vascularity and decalcification is well exemplified in these tumours, for one finds that the osteolytic

type is rapidly growing and very hyperæmic, whereas the osteoplastic type is more slow growing and less vascular. In the former type the increased vascularization is seen not only in the tumour itself but also in neighbouring bony parts. This parasarcomatous hyperæmia would account for the appearance presented in some cases where the bone beyond the limits of the tumour itself is seen to be markedly decalcified. So extensive may be the decalcification that inspection of the radiograph suggests extensive destruction of the bone. In a case of this type (*Fig. 315*) treated by X rays combined with colloidal lead, the bone appeared under treatment to reorganize almost completely. Presumably the diminution in the blood-supply resulting from the treatment gave rise to recalcification of the previously decalcified area.



FIG. 315.—Sarcoma of the scapula showing recalcification under treatment by X rays and colloidal lead. A, Nov. 8, 1923. B, Feb. 4, 1924.

When treatment was suspended, further decalcification took place, with subsequent recalcification when further treatment was given. The patient eventually died from metastases elsewhere.

If in the presence of generalized decalcification one fragment of bone has retained its normal calcium content, the radiologist can say with certainty that the fragment is avascular. It is this fact which enables him to report the presence of a sequestrum in osteomyelitis. But the sequestration of bone may be the result of trauma and not of infection. The carpal scaphoid bone is supplied with blood by a vessel which enters at the tubercle, and a fracture through the waist of the scaphoid will completely cut off the blood-supply of the proximal half of the bone. In such a case, although every other carpal

FIG.
316.



FIG. 316.—Fracture of the carpal scaphoid with evidence of avascularity of the proximal half of the bone. The proximal half of the scaphoid is of normal density (cf. upper shafts of radius and ulna). Unlike the distal half and the other carpal and metacarpal bones, it has not undergone decalcification. It is therefore avascular.

FIG. 317.—Fracture of the neck of the astragalus and of the internal malleolus. The body of the astragalus and the internal malleolus having retained their full calcium content must be avascular. This implies that there has been dislocation of the body of the astragalus which is now reduced.

FIG.
317.



bone may decalcify, the proximal half of the scaphoid remains normally dense (*Fig. 316*). If in another case there is normal density of the semilunar as well as of the proximal half of the scaphoid, the other bones being decalcified, the radiologist can say with certainty that there has been a dislocation of the semilunar and half scaphoid, although the dislocation may now be reduced. Similarly, if the astragalus shows normal density but every other adjacent bone shows decalcification, the radiologist knows that the astragalus is avascular and that it has been completely dislocated and its ligamentous attachments torn, although the bone may subsequently have been replaced (*Fig. 317*).

III. THE CLINICAL SIGNIFICANCE OF HYPERÆMIC DECALCIFICATION OF BONE.

Delayed Union of Fractures.—The decalcification of disuse frequently accounts for delay in the consolidation of fractures. It is well recognized that delayed union of leg fractures may be cured by simple weight-bearing in an ambulatory splint or plaster. The treatment has done nothing more than to balance the functional activity of the limb with the arterial blood-supply, so preventing further disuse decalcification and leading to recalcification. On this pathological fact is based the principle of early functional activity in the treatment of all fractures.³² It is this fact which explains the greater success of the dorsal plaster cast in securing bony union of fractures of the carpal scaphoid than the palmar cock-up splint which interferes with function.⁵¹

Non-union of Fractures.—It may be categorically laid down, contrary as it may be to existing teaching, that inadequate immobilization is the *only* factor of importance in establishing non-union. Before a fracture can unite, capillary blood-vessels and a continuous mass of young connective tissue must bridge the gap between the fragments. Not until the initial traumatic hyperæmia has subsided can the calcium salts be redeposited in the young connective tissue to form callus. When finally with increasing fibrosis the local blood-supply becomes impaired, the callus joining the two fragments undergoes dense calcification and sclerosis, and union is consolidated.

If, on the other hand, the fragments are imperfectly immobilized, the constant shearing and twisting strains rupture the capillaries between the fragments, and interrupt the continuity of the developing connective tissue. Moreover, the repeated traumatization gives rise to constantly recurring hyperæmia, and more and more of the bone-ends undergoes decalcification. Ultimately a linear fracture is represented by a cavity, the two bone-ends showing concave surfaces. When finally the hyperæmia does subside, there is no continuous mass of callus which can be recalcified, and sclerosis is observed in the plaque of bone across the concave bone-ends. Non-union is now established.

This sequence of events may constantly be seen in fractures of the scaphoid and fractures of the neck of the femur which have been imperfectly immobilized (*Figs. 318, 319*). Moreover, it is of significance that the fractures of the skeleton which have been the most common sites of non-union, are



FIG. 318.—A, Ununited fracture of scaphoid of six months' duration. B, By no treatment other than immobilization for many months, the fracture has united by bone; the traumatic hyperæmic decalcification has been arrested and the cavity has recalcified.



FIG. 319.—A, Ununited fracture of femur neck. Six months after injury. The patient has been in bed throughout. The continued traumatic hyperæmia from inadequate immobilization has caused decalcification of the neck. (Dr. J. H. Mather.) B, Similar fracture of neck of femur. Six months after injury. This patient has also been in bed moving his limb throughout, but the fracture has been immobilized by a stainless Smith Peterson nail, and no decalcification has occurred.

precisely those which are most difficult to immobilize completely.* Furthermore, in fractures of the true neck of the femur the percentage of cases securing bony union is directly proportional to the completeness of the immobilization. With no immobilization, not one unites by bone; with a Whitman plaster, which immobilizes to some extent but does not completely prevent shearing and rotative strains, about 50 per cent unite; with a Smith



FIG. 320.—A, Compound fracture of shafts of both leg bones treated by Winnett Orr plaster. There is extensive decalcification owing to traumatic and infective hyperæmia. B, Same case ten months later. There has been no treatment other than continued immobilization. As the traumatic and infective hyperæmia subsided the 'cavity' reossified.

Peterson nail⁵⁰ so well placed that immobility is absolute, practically every case unites by bone (*Fig. 319*).

In the infected compound fracture there is still more decalcification, because the initial hyperæmia of trauma is perpetuated by the hyperæmia of

* It should not be necessary to point out the fallacy in the argument that because fractures of the ribs always unite some degree of mobility encourages union. Although the whole rib moves with every respiration, it is a composite movement in which both fragments move together; they do not move on each other. There is no fracture in the skeleton in which there is a greater protection from shearing strain.

infection. But decalcification only delays union and does not prevent it. The explanation of the frequency of non-union in infected fractures lies in the fact that the treatment of the infection has usually been allowed to interfere with the immobilization. The Winnett Orr treatment⁵³ of compound fractures has helped to solve the difficulty, not only by maintaining absolute immobilization despite the infection, but by minimizing the loss of calcium through minimizing the volume of discharge. When this method of treatment is adopted the fragments may be seen to recalcify as soon as the infection

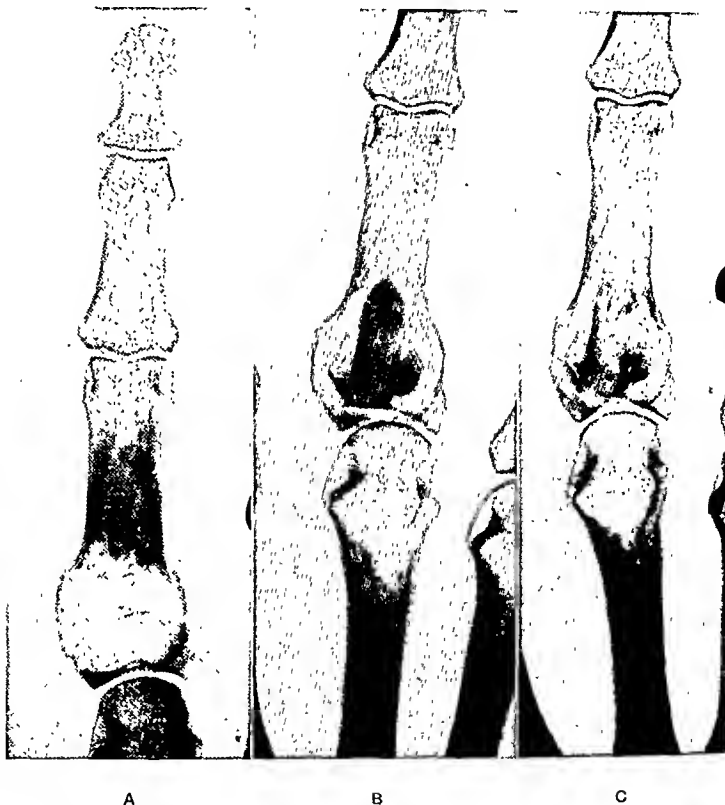


FIG. 321.—A, Chondroma of phalanx; B, One month after curettage and bone-grafting; C, Six months after bone-grafting. A month after operation the graft still has a greater density than the adjacent bone. It is therefore avascular and is dead.

becomes quiescent. Consolidation rapidly follows the removal of the last sequestrum and the healing of the last sinus, and even extensive gaps are filled and recalcified without bone-grafting proving necessary (*Fig. 320*).

The Transplantation of Bone.—When non-union of a fracture has become established with cavitation and with sclerosis of the bone-ends, a bone-grafting operation undoubtedly facilitates union. There has been considerable difference of opinion amongst surgeons as to whether the cells of the graft survive, or whether the graft dies and merely provides a framework for new bone formation. During the first day or two the undamaged cells on the surface

of the graft may perhaps grow and multiply. for even in the laboratory, with suitable hanging drop media. Fell⁵⁵⁻⁵⁹ has grown living cultures of bone. Nevertheless active growth of the graft cannot continue after the first few hours unless there is survival of its cells by revascularization. Radiographs taken at frequent intervals after operation show that the graft, so long as it remains visible in the films, has a greater density than the adjacent bone, which undergoes the decalcification of traumatic hyperæmia (*Fig. 321*). This in itself proves that the graft has remained avascular, and although its calcium store is utilized by continued surface erosion until it is entirely absorbed, the transplanted bone has never been within the circulation of its host.

A bone-grafting operation facilitates union of fractures in three ways: (1) The preparation of the bed of the graft breaks down the barrier of sclerosed bone and allows revascularization and mobilization of calcium salts: (2) The graft itself augments the local excess of calcium: and (3) The accurate fitting of the graft in its bed assists in securing the necessary immobilization. These three factors also apply when the bone-grafting operation is complicated by infection, and it is entirely wrong in these circumstances to remove the graft at an early stage. The transplanted bone is no more dead than in uninfected cases, and, provided that immobility is unceasingly maintained by a Winnett Orr plaster, the calcium salts liberated by the infective hyperæmia are utilized for the recalcification of the callus. In every sequestrum there is evidence of decalcification in the erosion and pitting of the surface. The only difference between a bone-graft and a sequestrum is that in the former case the fragment is completely decalcified by the vascular young connective tissue, whereas in the latter case decalcification is ultimately limited by the fibrosis and avascular scar-tissue formation of the later stages of infection. When once the infection has reached this chronic stage the graft can be of no further assistance, for it cannot be further decalcified, and any fragments responsible for sinus formation may be removed. But in a definite proportion of cases sufficient calcium has already been mobilized and the fracture is united.

The Robertson Lavallo operation⁶⁰⁻⁷⁰ of drilling into the decalcified areas adjacent to the surface of a tuberculous joint and laying a fragment of bone in the drill hole is merely another illustration of the value of a transplanted fragment of bone as a source of calcium salts. The operation cannot be of value through the hyperæmia it induces, for the decalcification proves that the region is already hyperæmic. The 'regeneration' which is claimed is merely recalcification of bone which has not been destroyed but has been decalcified by hyperæmia.

In the obliteration of bone cysts and benign tumours of bone the implantation of a graft is a procedure which facilitates bone regeneration by providing a reservoir of calcium (*see Fig. 321*), and there is evidence that boiled bone, beef bone, ivory, or a paste of calcium salts is equally effective.

Kümmell's Disease.—If it be accepted that Kümmell's disease of the spine,^{71, 72} with absorption of the vertebral body progressing weeks and months after injury, can occur in the absence of any fracture, it is to the

hyperæmic decalcification of contusion of the vertebra that the condition must be ascribed. One of the authors has shown elsewhere^{73, 74} that extensive crush fractures can be spontaneously reduced by simple hyperextension movement, and even perfect X-rays may fail to show evidence of the fracture which earlier X-rays proved to be present. In such cases there will undoubtedly be hyperæmia of the vertebral body, and it is not necessary to assume a rather problematical 'contusion' localized to one body. In every fracture of the vertebral body the resulting hyperæmia leads to decalcification and absorption. This still further facilitates the collapse and wedging of the vertebra which follows the breaking up of the cancellous framework unless it



FIG. 322.—Spontaneous hyperæmic dislocation of the atlas with cord pressure and quadriplegia. The dislocation was reduced, the child survived, and the paraplegia recovered. Two years later there was pyuria and tubercle bacilluria, but there has been no evidence, radiological or clinical, of tuberculous disease of the atlas. (Dr. Emrys Jones.)

is adequately protected. For this reason the deformity after a relatively simple untreated crush fracture of the spine never attains its maximum until several months after injury.

Spontaneous Hyperæmic Dislocation of the Atlas.—It has been pointed out that hyperæmic decalcification of bone does not necessarily imply infection of the bone itself, but may result from the spreading hyperæmia of an adjacent inflammatory focus. This is most clearly illustrated by the disastrous consequences of nasopharyngeal infections and other infections at the base of the skull, when the hyperæmia extends sufficiently to involve the atlas.

So marked may be the decalcification that the attachments of the transverse ligament are loosened and the anchorage of the odontoid to the atlas is impaired. Forward subluxation or dislocation of the atlas may now arise spontaneously.

The 14 recorded cases and 2 personal cases were recently reviewed by one of the authors,⁸² and since that publication 4 further cases have been observed by him, and others personally communicated, bringing the total to 21. The condition occurs most commonly at the end of the first decade. The nature of the infection and its exact location is of no significance; it may be tuberculous, specific, or pyogenic; it may involve the tonsil, the nasopharynx, the cervical glands, the retropharyngeal cellular tissue, the mastoid process, or the occipital bone. The one factor common to all cases has been an infection sufficiently acute and sufficiently near the atlas to give rise to hyperæmia but not necessarily to infection of the bone. In the majority of cases the dislocation has been readily reduced and a relatively short period of immobilization has been adequate in securing complete recovery.

Three clinical types must be distinguished, the pathology being identical in all types and the clinical differentiation dependent upon the degree of displacement.

In the group which is the most common, the atlas is dislocated forwards and slightly rotated, sufficiently to produce torticollis, painful movement, and spasm of the deep cervical muscles. In

the second group, of which no case has hitherto been reported in the literature, the displacement is more marked and the spinal cord is compressed; the compression is incomplete so that the patient survives, but a quadriplegia results—in the case now reported the dislocation was reduced and the paralysis fully recovered (*Fig. 322*). In the third group the dislocation is so complete that the cervical cord is completely compressed and the patient dies suddenly; the cases of Greig and of Reid are of this type, and *Fig. 323* shows the post-mortem specimen of a similar case in the Liverpool University Museum.



FIG. 323.—Spontaneous hyperæmic dislocation of the atlas with cord pressure and death. There is hyperæmic decalcification of the odontoid but no evidence of tuberculous disease. (*Post-mortem Specimen, Liverpool University Pathological Museum.*)

SUMMARY OF 21 CASES OF SPONTANEOUS HYPEREMIC DISLOCATION OF THE ATLAS.

AUTHOR	AGE	SEX	CAUSE OF HYPEREMIA
Dislocation without Complication:—			
Gillette ⁷⁵	2	?	Tonsillitis
Swanberg ⁷⁶	22	M.	Tonsillitis
Grisel ⁷⁷	8	F.	Tonsillitis
Grisel	9	F.	Nasopharyngitis
Desfosses ⁷⁸	7	F.	Nasopharyngitis
Berkheiser ⁷⁹	8	F.	?
Berkheiser	10	F.	Scarlet fever (? with pharyngitis)
Berkheiser	9	M.	'Cold in head'
Berkheiser	11	M.	'Sore throat'
Berkheiser	9	F.	Acute influenza
Watson Jones ⁸²	9	M.	Acute mastoid disease
Watson Jones	2	M.	Nasopharyngitis
Watson Jones*	17	M.	Osteomyelitis of occipital bone
Watson Jones*	11	F.	Pharyngitis and bronchitis
Fitzwilliams*	10	M.	Tonsillitis and cervical adenitis
Fitzwilliams*	15	M.	? Acute rheumatism
Dislocation with Quadriplegia:—			
Watson Jones*	7	M.	T.B. cervical glands
Dislocation with Sudden Death:—			
Chas. Bell ⁸⁰	?	F.	Syphilitic ulceration of pharynx
Greig ⁸¹	16	F.	Retropharyngeal abscess
Reid	16	F.	T.B. cervical glands
Watson Jones*	History	unknown	

* Not previously recorded.

Recumbency, Decalcification, and Nephrolithiasis.—Patients whose treatment demands prolonged recumbency and immobility frequently develop large numbers of calculi in the renal pelvis and ureter. So common is the complication and so extensive the calculus formation that the possibility of coincidence is excluded. Very frequently it is observed in open-air hospitals where cases of tuberculosis of the spine or of the hip-joint are immobilized for many months by spinal frames. But it is not confined to sanatorium patients, and cannot be explained by the hypervitaminosis of heliotherapy. Moreover, it is not confined to tuberculous patients, and even when there is tuberculous disease of bone there is no evidence of similar infection of the kidney. It may occur after fractures of the neck of the femur, and was seen in military hospitals in cases of gunshot wound with infected fracture of the thigh.⁸³⁻⁸⁹ Furthermore, calculi sometimes develop within some months of a severe debilitating disease such as pneumonia or empyema, where there is no primary bony abnormality.

The factor common to all cases is recumbency and general immobility. We have shown that immobility causes local decalcification of the bones, and the evidence of X rays shows that generalized disuse depletes the calcium store of the whole skeleton. Moreover, in the majority of cases, to the general decalcification of disuse is added the local decalcification of the disease. We know that the kidney has a high phosphatase activity, and in all of these



FIG. 324.—Nephrolithiasis following hypercalcemia of hyperparathyroidism.
(*Dr. Baker Bates and Dr. R. W. Gemmell.*)



FIG. 325.—Vesical calculi in Paget's disease.

eases the deposit suggests the precipitation of soluble salts through supersaturation, for not uncommonly the calculi disappear when activity is resumed. There would appear, therefore, to be little doubt that the nephrolithiasis is the result of skeletal decalcification and is comparable with the nephrolithiasis of hyperparathyroidism (*Fig. 324*). Amorphous vesical calculus formation in Paget's disease (*Fig. 325*) may be accepted as a similar type of calcium deposition in the urinary tract.

Despite the fact that the calculi may disappear with the resumption of activity, the dangers of this complication are not to be under-estimated. Quite apart from the risk of renal inefficiency due to obstruction, the prolonged excessive excretion of calcium in itself may lead to renal cellular degeneration and retention of non-protein nitrogen.³⁰ Frame patients should be taught regular exercises for those parts which need not be immobilized, the extent and duration of immobility should be minimized as far as possible, and the fluid intake should be increased throughout the period of recumbent treatment.

IV. THE CLINICAL SIGNIFICANCE OF ISCHÆMIC CALCIFICATION OF BONE.

The recovery of bone infections and the repair of bony injuries are characterized pathologically by fibrosis and scar-tissue formation with diminution of the blood-supply. The initial decalcification of hyperæmia is therefore superseded by the recalcification and sclerosis of reduced blood-supply. The hard solid bones of chronic osteomyelitis and the dense bone in the region of a well consolidated fracture are evidence of this.

Metastatic Malignant Disease of Bone.—The distinction between osteolytic and osteoplastic types of bone sarcoma, depending on the vascularity of the tumour, has already been discussed. Similar differences may be observed in the secondary bone deposits of carcinomata, and the most striking example is seen in the secondaries of carcinoma of the prostate. These diffuse deposits grow slowly, and are relatively avascular, with none of the power of invoking multiplication of neighbouring capillaries such as is constantly seen in the sarcomata. Unlike the rapidly growing deposits of breast carcinoma which lead to focal destruction, there is a slow and gradual infiltration of the whole of a vertebral body so that the vascular cancellous bone is replaced by relatively avascular osteoid tissue which ultimately becomes the site of dense calcification.

The Fragility of Calcified Bone.—The frequency of spontaneous fracture in bone which is apparently sclerosed, hard, and dense has not been capable of easy explanation. The significant point is that a dense X-ray shadow does not necessarily imply a hard bone. Marble and chalk cast a shadow of similar density,¹⁰² but the fragility is very different. The resistance of the skeletal framework to injury depends on the resilience of the normal lamellated bone and not on the degree of calcification. When it is recognized that the increased laying down of calcium is evidence of diminished blood-supply and reduced vitality, the abnormal fragility is readily understood.

In Paget's disease, one of the authors has shown⁹⁰ that fractures due to trivial injury are seen in the earlier stages of fibrous metaplasia where the bone shows granular calcification. They are not encountered in the later stages where calcification has progressed to re-ossification in the form of well-defined lamellæ or strands of new bone.

In syphilitic osteitis where there is endarteritis obliterans the characteristic bone change is sclerosis with an abnormally dense X-ray shadow. *Fig. 326* is from a case of Charcot's disease of the ankle, and there is evidence of osteitis of the os calcis. The bone is abnormally dense and there is increased calcification, but it is soft and has bent beneath the body weight.



FIG. 326.—Charcot's disease of the ankle with involvement of the os calcis. Although there is increased calcification of the os calcis, the bone is abnormally fragile and has bent beneath the body weight.



FIG. 327.—Osteosclerosis fragilis generalisata (Albers-Schönberg's disease). Fractures have occurred in the femora where the calcification is greatest. The bone is abnormally fragile. Note the rings of calcified bone in the pelvis parallel with the epiphyseal lines.

bent beneath the body weight. The angle of the bone is lost, exactly as it is in crush fractures of the os calcis due to a fall on the heel.

It is in the rare disease osteosclerosis fragilis generalisata, first described by Albers-Schönberg,⁹¹⁻¹⁰² that the association of increased calcification with fragility is most conspicuous. The term 'marble bones' which has been applied to the disease is misleading. 'Chalky bones' would be a more accurate description, for, despite their apparent density, the bones are relatively soft, easily drilled, and liable to spontaneous fracture. In one case which has been under investigation (*Fig. 327*) every epiphysis is involved and the disorder was apparent from an early age. There is evidence of periodicity of the disease in the rings of

sclerosed bone alternating with rings of more normal bone lying parallel with the epiphysial lines and particularly evident in the pelvis. As yet no evidence of general vitamin or endocrine disturbance has been found, it has not been possible to estimate the phosphatase activity of an epiphysis, and the factors responsible for the epiphysial abnormality are not known. But whatever future investigations may show, it is significant that fractures occur in those regions of the bone which are most densely calcified.

Kienböck's Disease and Preiser's Disease (*Traumatic Sclerosis and Porosis of the Carpus*).—In 1910 Preiser¹⁰³ described five cases of wrist injury where an oval or circular area of decalcification appeared in the waist of the scaphoid, leading later to the appearance of a fracture through the decalcified area. Preiser believed that the fracture did not precede the decalcification, but that it was pathological in type, occurring spontaneously as its result. Haenisch¹⁰⁴ showed, however, that in Preiser's original films there was usually evidence of a fissured fracture through the waist of the bone. The frequency of crack fracture of the scaphoid is much greater than is usually recognized, but the injury is easily overlooked in radiographs. The fracture is frequently in an oblique plane which coincides neither with the antero-posterior nor with the lateral planes of the wrist. Radiographs taken accurately in these axes may show no evidence of it, although it is clearly visible in a 'three-quarter' oblique view. A slight break in continuity of the articular surface may be the only manifestation, and the magnification of a lens may be necessary to pick out the line of fracture running across the bone. In some cases a fissured fracture of the scaphoid is suspected, but only proved by taking further radiographs after an interval of two or three weeks, by which time hyperæmic decalcification of the fragments due to the continued trauma of movement has made the injury obvious.

In every case of fracture of the scaphoid, however slight the crack may appear to be, progressive decalcification is observed unless the fragments are immobilized, and it is the progressive nature of the decalcification which proves the pre-existing fracture. The local areas of decalcification which may be observed in any carpal bone following a contusion or a ligamentous tear rapidly disappear as function is resumed. Only if the initial trauma is perpetuated by the repeated trauma of movement of fractured fragments on each other, does the decalcification progress. 'Preiser's disease', therefore, is not a clinical entity; it is a fracture of the scaphoid inadequately immobilized. The so-called 'cyst of the scaphoid' is merely a stage in the development of an ununited fracture.

If at any time an unpadded dorsal plaster cast is applied so that the bone is immobilized while functional activity is continued, the rarefaction disappears, the bone recalcifies, and the fracture unites. When immobilization is instituted immediately after the injury, decalcification is not observed and the fracture unites in six to eight weeks. When immobilization is delayed until after the fragments are decalcified, several months of fixation may be necessary (*see Fig. 318*). In either event the wrist becomes radiologically and clinically indistinguishable from normal. There is no kind of justification for excision of the bone as advocated by Hirsch,¹⁰⁶ and physiotherapy as suggested by Buchman¹⁰⁷ will obviously increase the hyperæmia,

accelerate the decalcification, render non-union inevitable, and account for the osteo-arthritis of the wrist which he describes as the usual end-result of conservative treatment.

If the wrist is not immobilized, the constantly repeated trauma leads ultimately to reparative fibrosis with impairment of the blood-supply, and an area of sclerosis is observed round the margin of the decalcified zone. Whereas decalcification is the response of bone to the immediate hyperemia of a single injury, increased calcification is the response of bone to the delayed ischæmia of constantly repeated injury. Herein lies the explanation of Kienböck's disease of the semilunar.¹⁰⁵ The condition is not a complication of fracture or dislocation of the semilunar.* It is not seen after a single wrist injury, but occurs most commonly in the right wrist of miners, gardeners, and other labouring men who sustain frequently repeated occupational contusions of the joint. The semilunar occupies the key position of the carpal arch and is constantly subjected to compression forces. The immediate effect of these minor injuries is seen occasionally in small areas of traumatic hyperæmic decalcification; the cumulative delayed effect is seen in the ischæmic sclerosis of the bone known as Kienböck's disease. When the bone is removed it is found to be relatively bloodless, fragile, and mis-shapen. Microscopical examination shows areas of fibrosis and general necrosis of the bone.

In considering treatment it must be recognized that Kienböck's disease represents the end-result of a long-standing pathological process, and symptoms are due not merely to necrosis of the bone but to mechanical derangement of the joint owing to alteration in the shape of its articular surfaces. As would be expected, prolonged immobilization has no effect whatever on the sclerosis of the bone. Physiotherapeutic treatment and operative drilling of the bone with the object of revascularizing it have been attempted but without amelioration of the symptoms. The only prospect of relief is by excision of the bone, and although no wrist from which the semilunar has been removed recovers normal movement or strength, the joint becomes sufficiently stable and painless to enable moderately heavy work to be resumed.

Köhler's and Freiberg's Disease of the Metatarsal.—Köhler's disease of the metatarsal¹⁰⁶ (Panner's or Freiberg's disease) is exactly analogous to Kienböck's disease of the semilunar. There is evidence of impairment of the blood-supply not only in the increased density of the bone, but in the fragmentation and distortion of the metatarsal head which results in arthritic change in the metatarso-phalangeal joint. Excision of the head of the bone relieves the symptoms, and histological examination of the bone shows definite evidence of fibrosis and necrosis, confirming the fact that impairment of blood-supply is the underlying cause. The factors responsible for the interference with blood-supply are not yet clearly established, although the frequent association of the condition with congenital anomalies of the first metatarsal which increase the weight-bearing strain on the affected one, would point to repeated trauma.

* This fact has again been confirmed by Mr. Wainwright at the Liverpool Royal Infirmary, who followed up twenty-five cases of dislocation of the carpal semilunar bone several years after manipulative or operative reduction. In no case were the changes of Kienböck's disease found.

V. THE CLINICAL SIGNIFICANCE OF PATHOLOGICAL CALCIFICATION.

Supraspinatus Calcification.—It is now well known that the calcareous masses seen on the outer aspect of the shoulder-joint, which originally were thought to lie in the subdeltoid bursa,¹¹¹⁻¹¹⁶ have actually developed within the tendon of the supraspinatus. The amorphous calcium carbonate and phosphate forms a semi-liquid mass between the fibres of the tendon with no capsule or limiting membrane. There is neither

FIG. 328.—Calcification of the supraspinatus tendon.

clinical nor microscopic evidence "of infection, and the pathological features of this condition differ in no respect from those of calcification elsewhere. It is not remarkable that of all the tendons in the body it is the supraspinatus which most commonly undergoes this change, for it is the only

tendon in which there is normally constant compression against bone. Immediately above its insertion into the great tuberosity it occupies a vulnerable position, and impinges against the acromion with every abduction movement of the limb.

FIG. 329.—Calcification of the supraspinatus tendon. Before operation. Same case as Fig. 338 (ossification of the tendo Achillis). (Dr. Ransome's case.)

The effect of this bony compression is seen in the fairly common syndrome of chronic supraspinatus tendonitis. There is tenderness immediately above the great tuberosity, and the arc of movement



between 60° and 120° of abduction is painful. The radiograph may show no abnormality and the symptoms subside with rest. Occasionally, however, a case is seen in which the symptoms are much more acute, and a localized swelling of the tendon may be felt as a nodule beneath the deltoid. The radiograph now shows that the fibrosis of constant trauma has resulted in calcification (*Fig. 328*). There is no room for expansion of the tendon between the tuberosity and the acromion, so that the two positions of comfort are with the arm by the side, or in hyper-abduction so that the tendon lies wholly beneath the acromio-clavicular joint and not in contact with the outer margin of the acromion. In a severe case the support of a sling is intolerable, and the arm must hang by the side with the elbow extended in order that the weight of the limb may increase as far as possible the space between the humerus and the acromion (*Fig. 329*).

Since the pain is of purely mechanical origin, the most rapid relief is secured by incising and evacuating the mass. A short incision is all that is required, and it is unnecessary to do more than to relieve the tension. If the deposit is sufficiently fluid to be withdrawn through an aspirating needle, the relief is equally complete and permanent. In less advanced cases the symptoms subside spontaneously as the volume of the deposit is reduced by desiccation. Moreover, an irritative hyperæmia may supervene and lead to the re-absorption of small deposits.

Calcification of the Hæmatoma and the Cavernous Angioma.

—A subperiosteal hæmatoma

of bone is in direct association with an extensive store of calcium which is mobilized by the hyperæmia of the injury. From the earliest stages ossification is observed round the periphery of the blood-clot, gradually spreading throughout it. On the other hand, if a hæmatoma develops in soft tissues distant from bone, the only available source of calcium is through the bloodstream. No change occurs until the hæmatoma is organized and fibrous tissue has developed. The avascular tissue may now undergo calcification.



FIG. 330.—Cavernous angioma of forearm with calcification of thrombosed blood. X-ray showing diagnostic appearances.

The sequence of events may be observed in the blood-clot within a vein which by calcification gives rise to a phlebolith. Although less commonly recognized, it is exactly the same process which accounts for the calcareous nodules in cavernous angiomata of muscles (*Fig. 330*). The nodules are shown radiographically as concentric rings varying in size from 1 mm. to 1 cm. in diameter. The calcareous deposit is not within the walls of the blood-vessels, but occurs only in the fibrous masses of organizing blood-clot. This was suggested by Ruggles,¹¹⁷ who first drew attention to the condition in 1916,

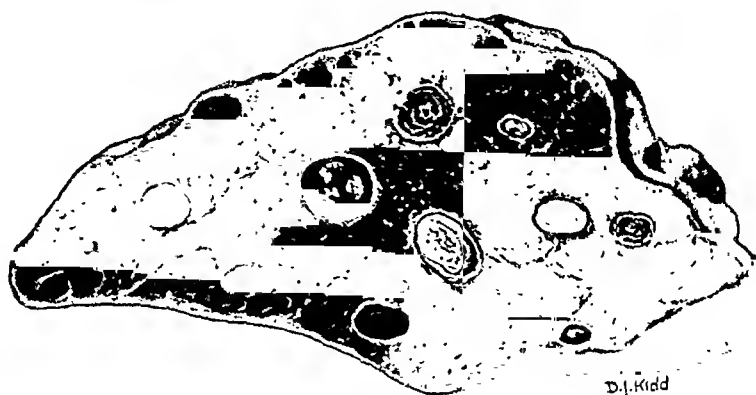


FIG. 331.—Cavernous angioma with calcification. Same case as *Fig. 330*.

and has been confirmed by examination of two recent cases (*Fig. 331*). As more blood-clot becomes organized, further concentric rings are added to the original nodule. If the shadows are seen in the regions where phleboliths do not occur, the appearances are diagnostic of the cavernous angioma.

Calcification in Raynaud's Disease.—Calcification in the skin and subcutaneous tissues of the fingers of patients suffering from Raynaud's disease was first described in 1911 by Thibierge and Weissenbach.¹²⁶ The concretions show a similar chemical constitution to pathological calcareous deposits elsewhere, and there is no alteration in the serum calcium concentration. Moreover, the calcification usually occurs after the phase of vasoconstriction with 'local asphyxia', and microscopic examination of the tissues shows fibrosis. The calcification occurs most constantly in the terminal phalanges, where there is normally a preponderance of fibrous tissue and where the impairment of vascularity will be most marked (*Fig. 332*). But deposits may also be seen in the subcutaneous tissues of the forearm (*Fig. 333*), and the radiographic features are very similar to those of the calcified cavernous angioma. Moreover, Weissenbach has described, and we have confirmed, the occurrence of multiple telangiectases in the skin and mucous membranes of cases of Raynaud's disease exhibiting calcinosis. It would appear, therefore, that in Raynaud's disease we have yet another example of calcification in fibrous tissue of impaired vascularity, and that the fibrosis may be increased by the organization of thrombi in small angiomata.



FIG. 332.—Calcification in Raynaud's disease.

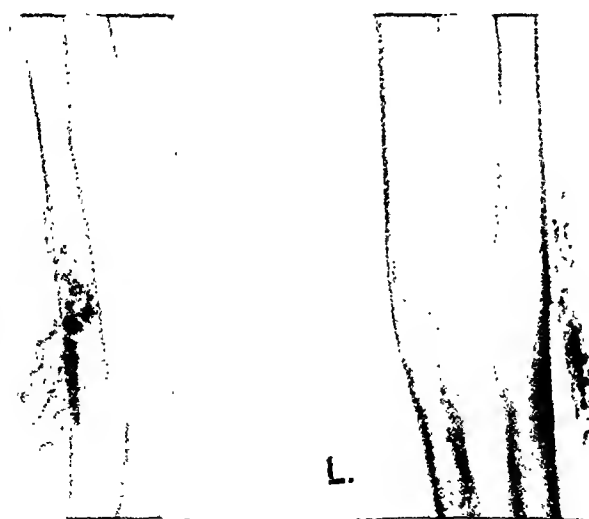


FIG. 333.—Calcification of forearm in Raynaud's disease.
Same case as *Fig. 332*.

Calcification of Intervertebral Discs.—Calcification in the nucleus pulposus of the intervertebral disc was first described by Calvé and Galland,¹³² and many cases have since been recorded.¹³³⁻¹⁴⁶ The deposit corresponds in shape to the nucleus pulposus and occupies the central part of the space between two vertebral bodies (*Fig. 334*). It is significant that this part of the disc is

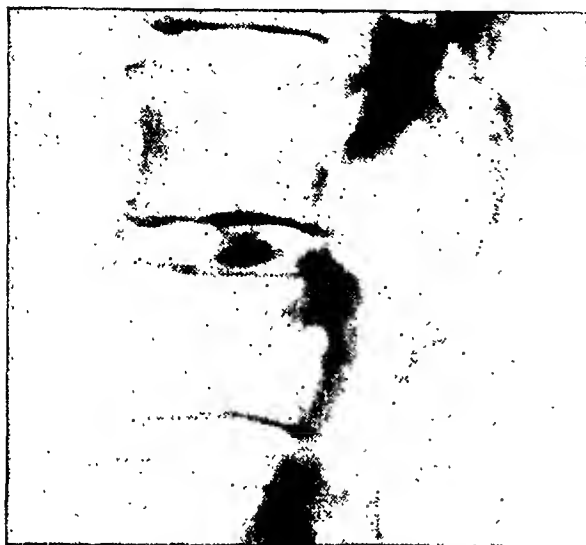


FIG. 334.—Calcification of intervertebral disc. There is calcification of the nucleus pulposus of one disc, and peripheral calcification of several discs.

avascular and normally of low metabolism, and that calcification is seen most often at the dorsolumbar junction of the spine, where the minor strains of movement and of weight-bearing are concentrated. As with pathological calcification elsewhere, so soon as the deposit is sufficiently extensive to account for increased tension, pain is produced. Since the nucleus is avascular, these central deposits remain an amorphous mass of calcium and do not undergo conversion to bone unless vascularization takes place through destruction of the protective cartilaginous discs. On the other

hand, Israelski¹⁴⁰ has described calcification in the peripheral part of an intervertebral disc immediately adjacent to the anterior common ligament. This part of the disc is capable of revascularization, and ossification is therefore frequently observed.

Calcification of Semilunar Cartilages.—In 1927 Mandl¹⁴⁸ pointed out that microscopic deposits of calcium were sometimes found in the semilunar cartilages of the knee-joint, and eight cases have since been published in German literature of calcification of the cartilages diagnosed during life by radiographic examination.¹⁴⁸⁻¹⁵⁵ The average age was over 45, but there has been no osteo-arthritic change in the joints, and not infrequently the disorder was entirely symptomless. Frequently both cartilages were involved and the calcium was diffusely distributed. In one case microscopic examination showed "depletion of the cartilage cells with predominance of fibrous tissue". One patient also showed pathological calcification of the popliteal artery and of the peripheral part of an intervertebral disc.¹⁵² There can be no doubt, therefore, that calcification of the semilunar cartilages is evidence of a degenerative change in a relatively avascular fibrocartilaginous tissue. Since the deposit is usually diffuse so that the tension is not raised, the condition is not as a rule of great clinical significance, but it has an important bearing on ossification of the semilunar cartilage shortly to be described.

VI. THE CLINICAL SIGNIFICANCE OF PATHOLOGICAL OSSIFICATION.

In the clinical applications of pathological ossification two general types of bone formation must be distinguished: (1) Subperiosteal ossification; and (2) Heterotopic ossification. The first group includes not only the ossifying subperiosteal hæmatoma of the shaft of a long bone, but all forms of traumatic 'myositis ossificans'. The second group includes the localized formation of bone in scars and tendons, and the generalized form of fibrositis ossificans progressiva.

Subperiosteal Ossification and 'Myositis Ossificans'.—The periosteum is a fibrous membrane so closely applied to bone that when it is elevated it frequently carries with it bone cells. The membrane itself, however, is to be regarded as the limiting membrane of bone, and if the periosteum is raised, new bone is formed within the new limits of the periosteum. The periosteal elevation may be gradual and progressive from the traction of an active muscle. The 'rider's bone' and the spurs of the olecranon and of the os calcis are classical examples. The gluteal ridge, the linea aspera, and other normal bone ridges arise in the same way, and Greig¹⁰ has shown how œdema of the periosteum will accentuate these muscle markings by facilitating periosteal elevation. Herein lies the explanation of the roughening of the fibula which occurs after osteomyelitis of the tibia, and the roughening of



FIG. 335.—Ossification of traumatic subperiosteal hæmatoma of the shaft of the femur.

the shaft of any bone which has been fractured, especially when abnormal muscle strains are produced by malunion. On the other hand, the periosteum may be suddenly elevated by the hæmorrhage of scurvy or of injury. A hæmatoma limited by the displaced periosteum is in direct association with a tremendous store of calcium which is mobilized by the hyperæmia of the trauma, and ossification rapidly spreads throughout the mass of blood-clot. In the adult the condition is most commonly seen in the shaft of the femur after a direct kick (Fig. 335). The size of the traumatic osteoma is reduced

by spontaneous absorption of the hæmatoma, and may be minimized by aspiration in the early stages.

Several years ago one of the authors^{156, 157} showed that the condition known as 'myositis ossificans' or 'tendonitis ossificans' was an exactly similar type of pathological ossification. The periosteum is elevated by the avulsion from bone of a muscle attachment, and ossification occurs within the subperiosteal hæmatoma. It is most common in children because the periosteum is less firmly attached to bone in the child than in the adult. It is most common at the elbow because this is the joint most commonly dislocated, and it is a joint which cannot be dislocated without the avulsion of muscles.



FIG. 336.—Avulsion of the brachialis anticus and common flexor muscles, with ossification of the subperiosteal hæmatoma. The avulsion produced by dislocation of the elbow has been perpetuated by passive extension movements of the joint. (Dr. J. H. Mather.)

In adequately treated cases the disorder is never seen, because if the dislocation is reduced at once, the periosteum is replaced and becomes re-attached. But if reduction is delayed, and particularly if passive movements are practised, the periosteum is re-displaced, and is continually dragged farther away from the bone. The movement which is most commonly forced in injudicious treatment is extension, and, as would be expected, the periosteal displacement and new bone formation is seen at the attachment of the brachialis anticus to the ulna, and of the common flexors of the forearm to the humerus. A spur of bone may develop at both sites in the same case, and the subperiosteal hæmatomata may communicate with each other and produce a continuous bridge of bone (Fig. 336).



FIG. 337.—Avulsion of the quadriceps with ossification of the subperiosteal hæmatoma. The knee-joint has not been immobilized, so that the new bone has remained fragmented and separated from the patella.

Similarly at the knee-joint, the quadriceps may be avulsed from the patella. If the muscle is stitched back, no new bone formation is seen. If it is not operated upon, the tonic contraction of the quadriceps maintains the periosteal displacement and bone develops in the subperiosteal hematoma above the patella. The new bone fuses with the patella if the joint is immobilized in extension, but it remains fragmented and is more diffuse if movements are carried out (*Fig. 337*).

Similarly, the ligamentum patellæ may be avulsed from the lower pole of the patella, and new bone is laid down unless the ligament is re-attached by operation.

The same sequence of events may occur at the hip-joint or at the shoulder-joint after traumatic dislocation where reduction is delayed or early passive movement practised, the periosteum in these cases being avulsed by the capsular ligaments. Bone spurs developing immediately in front of the ankle-joint are of exactly similar type and are due to avulsion of the anterior capsule. The term 'myositis ossificans' should not be applied to these cases. There is no myositis. The bone is not formed in muscle or in tendon. It is the subperiosteal ossification of avulsed muscles.

Fibrositis Ossificans Progressiva.—The use of the term 'myositis ossificans' should be confined to cases of the progressive disease, which differs etiologically, pathologically, and clinically from the traumatic condition we have discussed. Greig¹⁰ has suggested the more accurate nomenclature "fibrositis ossificans progressiva", for even in this condition new bone is formed, not in muscles, but in the fibrous-tissue planes of tendons and muscles. The fibrous tissue undergoes de-differentiation to the embryonic state and is built up again as bone. The factors determining this de-differentiation are unknown, but there is definite evidence that the disorder is of congenital origin.

Ossification of the Tendo Achillis.—True heterotopic ossification may result from the revascularization of pathological calcareous deposits. In the tendo Achillis it would be possible for pathological calcification to occur without giving rise to symptoms, because the mechanical factors which account for pain in calcification of the supraspinatus tendon are absent. This may explain the fact that although eight cases of ossification of the tendo Achillis have been described,¹⁵⁸⁻¹⁶¹ the earlier stage of amorphous calcification has not been observed. In many of the recorded cases of heterotopic ossification of the tendo Achillis, and in the two further cases now recorded (*Fig. 338*), the diagnosis has been established twenty years or more after subcutaneous tenotomy. It may be accepted, therefore, that there has been fibrosis of a tendon of low vascularity which could produce a calcareous deposit, and that after a long interval of time the irritative effect of the deposit has led to revascularization with the development of bone.

An attempt was made by Macdonald at the Shropshire Orthopædic Hospital, and Sayle Creer at the Liverpool Royal Infirmary, to estimate the frequency of ossification as a complication of tenotomy of the tendo Achillis. but although fifty patients who had undergone tenotomy were X-rayed, none showed calcification or ossification. It was found impossible to trace the older patients, and the average interval of time which had elapsed since tenotomy was less than ten years.

As a general rule the ossification has not been associated with serious symptoms, but in Mallinson's case¹⁶³ the resulting diminution in the elasticity of the tendon predisposed to spontaneous rupture necessitating operative suture.

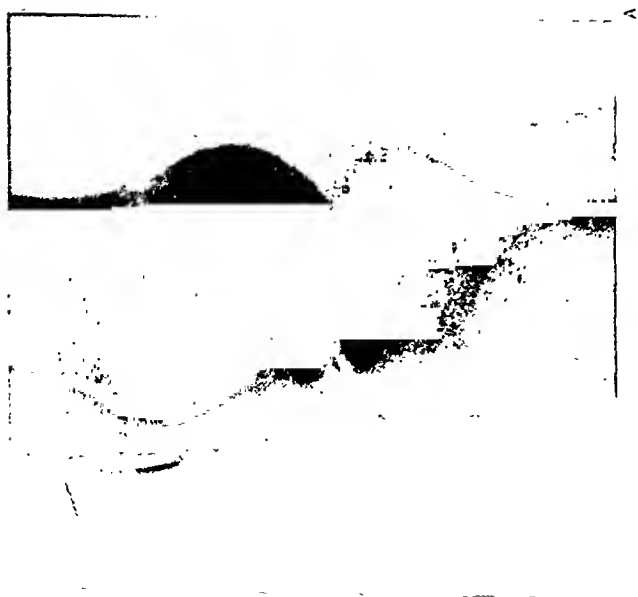


FIG. 338.—Ossification of the tendo Achillis. Twenty years after subcutaneous tenotomy. Same case as Fig. 329 (calcification of supraspinatus tendon).

Ossification of the Semilunar Cartilages.—Although several cases of calcification of the semilunar cartilages have been reported (p. 488), we have been unable to find any reference in the literature to ossification of the cartilage, and we believe that two cases are here recorded for the first time (Fig. 339). In both cases the external cartilage was involved, the bone deposits varying in size from that of a pea to that of a large hazel-nut. Although in the first case one deposit was in the anterior cornu and extended into the fibrous attachment of the cartilage to the tibia, there was no continuity with normal bone; moreover, in the other case two discrete bony deposits, one in the anterior third and one in the posterior third.



FIG. 339. Ossification of external semilunar cartilage. A, Radiograph of knee-joint; B, Radiograph of cartilage after excision.



B

were entirely surrounded by normal cartilage (*Fig. 340*). The condition is therefore a true example of heterotopic bone formation. In view of the recorded cases of pathological calcification of the cartilages there is no difficulty in applying the theory of etiology which has been applied to heterotopic ossification elsewhere.

In neither case was there an associated arthritic change, but both patients presented symptoms of internal derangement—weakness, recurring effusion, ‘giving way’ of the joint, and pain in the outer compartment. In the case in which there was a large deposit in the anterior third of the cartilage the terminal 15° of extension were limited by an elastic

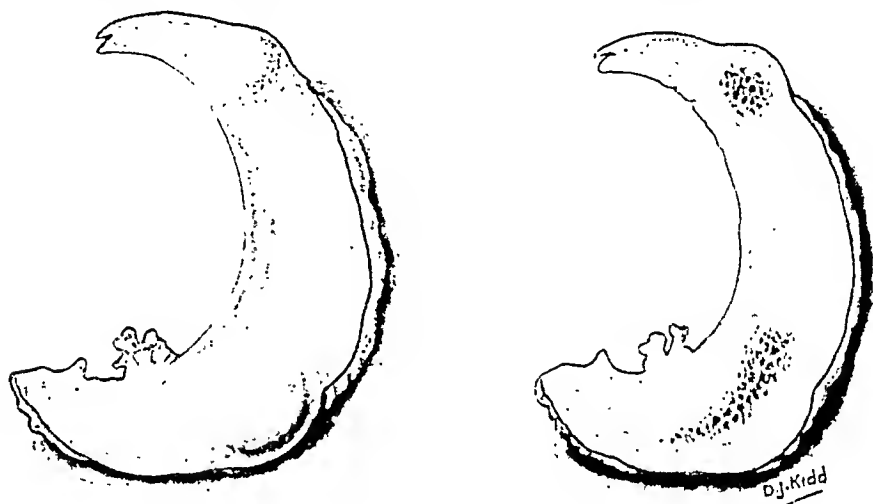


FIG. 340.—Ossification of external semilunar cartilage. Specimen removed from second case.

resistance suggestive of an unreduced bucket-handle cartilage tear. In both cases the pre-operative clinical and X-ray diagnosis was “loose bodies in the knee-joint”. In the first it was obvious as soon as the joint was opened that there was a tumour of the cartilage, and the operation presented no difficulty. In the second case a prolonged search for the expected loose body was made before the possibility of ossification of the cartilage was recalled. Removal of the cartilage relieved the symptoms in both cases. Histological examination of the cartilage shows fully developed bone with Haversian systems.

SUMMARY.

An attempt has been made to correlate the pathological, biochemical, and clinical features of normal and abnormal calcification, decalcification, and ossification.

Pathological Considerations.—

1. Bone undergoes decalcification if the blood-supply is increased, and increased calcification if the blood-supply is decreased.

2. Pathological calcification is observed in any mesenchymatous tissue of low metabolism when the vascularity is further reduced by the fibrosis of trauma or of infection.

3. Bone may form in any region where there are fibroblasts, excess of calcium, and an adequate blood-supply.

Radiological Applications. —

1. The radiologist must interpret decalcification of bone as evidence of hyperæmia, and increased calcification as evidence of ischæmia. If in the presence of decalcification of adjacent bones one fragment retains its original calcium content, the fragment is avascular. This may prove dislocation which is now reduced (e.g., astragalus, semilunar, scaphoid).

2. Hyperæmic decalcification must be distinguished from destruction. Decalcified bone adjacent to an infective focus may appear to be destroyed though actually normal. In tuberculous disease the area of destruction is much less than the area of decalcification. In osteolytic sarcomata the tumour is less extensive than X-rays suggest. Apparent regeneration after treatment is due to recalcification.

Hyperæmic Decalcification of Bone.---

1. Non-union of fractures is due to traumatic hyperæmic decalcification from inadequate immobilization. Compound fractures, although more decalcified owing to infective hyperæmia, may still unite if immobilized.

2. A bone-graft never becomes vascularized. It is a local excess of calcium which assists in the repair of ununited fractures, tuberculous osteitis, and bone cysts.

3. Three degrees of spontaneous hyperæmic dislocation of the atlas may occur: (a) without complication, (b) with paraplegia, (c) with sudden death.

4. Nephrolithiasis may result from generalized disuse and recumbency in fractures, tuberculous joints, pneumonia, etc., owing to excessive excretion of calcium. It is similar to the renal calculi of osteitis fibrosa and the vesical calculi of osteitis deformans.

Ischæmic Sclerosis of Bone.—

1. Increased calcification of bone is evidence of diminished vitality. The elasticity is reduced and the bone is abnormally fragile (Paget's disease, Albers-Schönberg's disease, Charcot's disease).

2. Kienböck's disease of the semilunar is due to impaired vascularity. The bone undergoes necrosis and must be excised. Preiser's disease of the scaphoid is not a clinical entity. It is a stage of non-union of fracture of the scaphoid and is cured by immobilization.

3. Panner's and Freiberg's disease of the metatarsal are due to impaired vascularity and are analogous to Kienböck's disease.

Pathological Calcification.—

1. Calcification of the supraspinatus tendon is due to the trauma of impingement of the tendon against bone. Pain is due to tension, and symptoms are relieved by any measure which reduces tension. Spontaneous absorption may occur.

2. The hæmatoma undergoes calcification when it is organized. The phlebolith, the calcified cavernous angioma, and the calcinosis of Raynaud's disease are of a similar type.

3. Calcification of the intervertebral disc and the semilunar cartilage are due to impairment of vascularity from repeated slight traumata.

Pathological Ossification.—

1. Whenever the periosteum is elevated, bone is found within the new limits of the periosteum (bone spurs, normal bone ridges, subperiosteal hematomata).

2. The subperiosteal ossification of avulsed muscles may occur at the elbow, knee, ankle, or shoulder. It is purely traumatic and can be prevented. The term 'myositis ossificans' should not be applied to these cases but should be reserved for the progressive disease.

3. Heterotopic ossification of tendon is most common in the tendo Achillis; in such cases tenotomy is frequently a causative factor.

4. Ossification may occur in the semilunar cartilages. The condition is difficult to differentiate radiologically from loose bodies in the knee-joint.

REFERENCES.

Theories of Pathological Calcification.—

- ¹ GRANDIS and MAININI, *Arch. ital. de Biol.*, 1900, i, 73.
- ² PFAUNDLER, *Jahrb. f. Kinderheilk.*, 1904, lx, 123.
- ³ PAULI and SAMEC, *Biochem. Zeits.*, 1909, xvii, 235.
- ⁴ HOFMEISTER, *Ergebn. d. Physiol.*, 1910, x, 429.
- ⁵ WELLS, *Arch. of Internal Med.*, 1911, vii, 721.
- ⁶ FREUDENBERG and GYONGY, *Biochem. Zeits.*, 1920, ex, 299.
- ⁷ WATT, *Arch. of Surg.*, 1925, x, 983.
- ⁸ LERICHE and POLICARD, *La Physiologie normale et pathologique de l'Os*, 1926. Paris.
- ⁹ LERICHE and POLICARD, *Physiologie pathologique chirurgicale*, 1930. Paris.
- ¹⁰ GREIG, *Surgical Pathology of Bone*, 1931. Edinburgh.

The Chemical Constitution of the Bone Salt.—

- ¹¹ GASSMANN, *Zeits. Physiol. Chem.*, 1910, lxx, 161.
- ¹² BASSETT, *Jour. of Chem. Soc.*, 1917, cxi, 620.
- ¹³ DE JONG, *Rec. Trav. chim.*, 1926, xlv, 445.
- ¹⁴ TAYLOR and SHEARD, *Jour. of Biol. Chem.*, 1929, lxxxi, 479.
- ¹⁵ ROSEBERRY, HASTINGS, and MORSE, *Ibid.*, 1931, xc, 395.
- ¹⁶ MORGULIS, *Ibid.*, 1931, xciii, 455.
- ¹⁷ BOGERT and HASTINGS, *Ibid.*, 1931, xciv, 473.

The Phosphatase Activity of Tissues.—

- ¹⁸ ROBISON, *Biochem. Jour.*, 1923, xvii, 286.
- ¹⁹ ROBISON and SOAMES, *Ibid.*, 1924, xviii, 740.
- ²⁰ MARTLAND and ROBISON, *Ibid.*, 1925, xix, 1354.
- ²¹ ROBISON, *The Significance of Phosphoric Esters in Metabolism*, 1932. New York.
- ²² HUGGINS, *Biochem. Jour.*, 1931, xxv, 728.
- ²³ HUGGINS, *Arch. of Surg.*, 1931, xxii, 377.
- ²⁴ MARTLAND, *Biochem. Jour.*, 1925, xix, 117.

Metastatic Calcification in Hypercalcaemia.—

- ²⁵ HUEPER, *Arch. Pathol. and Lab. Med.*, 1927, iii, 14.
- ²⁶ DAWSON and STRUTHERS, *Edin. Med. Jour.*, 1923, xxx, 421.
- ²⁷ BULGER DIXON, and BARR, *Jour. Clin. Invest.*, 1930, ix, 143.
- ²⁸ QUICK and HUNSBERGER, *Jour. Amer. Med. Assoc.*, 1931, xvi, 745.
- ²⁹ HUNTER, *Brit. Jour. Surg.*, 1931, xix, 203.
- ³⁰ HUNTER, *Lancet*, 1930, i, 897.

Calcification of Costal Cartilages.—

- ³¹ CHAUMET and ZAWADOWSKI, *Rev. de Tubercul.*, 1927, viii, 574.
- ³² RIST, GALLY, and TROCME, *Presse med.*, 1928, xxxvi, 641.

- ³³ RIEREL, *Amer. Jour. Roentgenol.*, 1929, xxi, 44.
- ³⁴ HILLY, *Zeits. f. d. ges. Anat.*, 1929, lxxxviii, 746.
- ³⁵ ERNST, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1929, xxxix, 185.

Calcification of Pineal Body.—

- ³⁶ BRONNER, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1926, xxxv, 277.
- ³⁷ CIGNOLINI, *Rev. franç. d'Endocrinol.*, 1927, v, 324.
- ³⁸ FAELLI, *Endokrinologie*, 1930, vii, 189.
- ³⁹ TOUSSAINT, *Arch. méd. belges*, 1930, lxxxi, 462.
- ⁴⁰ ELKINGTON, *Proc. Roy. Soc. Med.*, 1932, xxv, 1533.

Calcification of the Pericardium.—

- ⁴¹ CUTLER and SOMAN, *Amer. Jour. Roentgenol.*, 1924, xii, 312.
- ⁴² AMUNDSEN, *Acta. Radiol.*, 1923, ii, 38.
- ⁴³ CASE, *Jour. Amer. Med. Assoc.*, 1923, lxxx, 236.
- ⁴⁴ SCHOLZ, *Arch. of Internal Med.*, 1924, xxxiv, 32.
- ⁴⁵ JONES and ROBERTS, *Brit. Jour. Radiol.*, 1927, xxi, 167.

Calcification in Uterine Fibroids and Fibromata.—

- ⁴⁶ MATHER, *Brit. Jour. Radiol.*, 1927, xxxii, 257.
- ⁴⁷ ARENS and BLOOM, *Radiology*, 1928, x, 256.
- ⁴⁸ ALIX, *Jour. de Radiol. et d'Electrol.*, 1928, xii, 287.
- ⁴⁹ ARCHER and GREENBERG, *Amer. Jour. Obst. and Gynecol.*, 1931, xxi, 335.

Immobilization and Union in Fracture of Neck of Femur and Scaploid. -

- ⁵⁰ SMITH PETERSEN, *Arch. of Surg.*, 1931, xxiii, 715.
- ⁵¹ BÖHLER, *Treatment of Fractures*, 1929, Vienna.
- ⁵² WATSON JONES, *Brit. Med. Jour.*, 1932, i, 1073.

Treatment of Infected Fractures.—

- ⁵³ WINNETT ORR, *Osteomyelitis and Compound Fractures*, 1929, London.
- ⁵⁴ WATSON JONES, *Jour. Bone and Joint Surg.*, 1932, xiv, 609.

Osteogenesis in Vitro.—

- ⁵⁵ FELL, *Arch. f. exper. Zellforsch.*, 1928, vii, 70.
- ⁵⁶ FELL and ROBISON, *Biochem. Jour.*, 1929, xxiii, 767.
- ⁵⁷ FELL and ROBISON, *Ibid.*, 1930, xxiv, 1905.
- ⁵⁸ FELL, *Arch. f. exper. Zellforsch.*, 1931, xi, 245.
- ⁵⁹ FELL, *Jour. of Anat.*, 1932, lxxvi, 2, 157.

Robertson-Lavalle Operation for Tuberculous Joints.—

- ⁶⁰ ROBERTSON-LAVALLE, *Ann. de la Faculté de Méd. de Montevideo*, 1919.
- ⁶¹ ROBERTSON-LAVALLE, *Bull. et Mém. Soc. de Chir.*, 1923, xlv, 812.
- ⁶² ROBERTSON-LAVALLE, *Rev. d'Orthop.*, 1924, xi, 5.
- ⁶³ ROBERTSON-LAVALLE, *Arch. latino-amer. de Pédiat.*, 1925, Nov.
- ⁶⁴ RUIZ-MORENO, *Semana Méd.*, 1927, ii, 106.
- ⁶⁵ MOUCHET, *Congrès Chir.*, 1925, xxxiv, 276. Paris.
- ⁶⁶ OMBRÉDANNE, *Bull. et Mém. Soc. de Chir.*, 1926, lii, 955.
- ⁶⁷ SORREL, *Ibid.*, 1925, Dec.
- ⁶⁸ TAVERNIER, *Lyon chir.*, 1930, xxvii, 645.
- ⁶⁹ UFFREDUZZI and BIANCALANA, *Presse méd.*, 1931, xxxix, 242.
- ⁷⁰ VIGNARD, *Lyon. chir.*, 1927, xxiv, 576.

Kümmell's Disease of the Spine.—

- ⁷¹ KÜMMEL, *Deut. med. Woch.*, 1895, xxi, 280.
- ⁷² CALVÉ, *Jour. de Radiol. et d'Electrol.*, 1925, ix, 22.
- ⁷³ WATSON JONES, *Jour. Bone and Joint Surg.*, 1930, xiii, 383.
- ⁷⁴ WATSON JONES, *Brit. Med. Jour.*, 1931, i, 31.

Spontaneous Hyperæmic Dislocation of the Atlas.—

- ⁷⁵ GILLETTE, *N.Y. Med. Jour.*, 1896, lxiv, 158.
- ⁷⁶ SWANBERG, *Jour. Amer. Med. Assoc.*, 1919, lxxii, 107.
- ⁷⁷ GRISEL, *Presse méd.*, 1930, xxxviii, 50.
- ⁷⁸ DESFOSSÉS, *Ibid.*, 1930, xxxviii, 1179.
- ⁷⁹ BERKHUISER and SEIDLER, *Jour. Amer. Med. Assoc.*, 1931, xevi, 517.
- ⁸⁰ BELL, *Nervous System of the Human Body*, 1830, App. 127, 64.
- ⁸¹ GREIG, *Surgical Pathology of Bone*, 1931, 228. Edinburgh.
- ⁸² WATSON JONES, *Proc. Roy. Soc. Med.*, 1932, xxv, 586.

Renal Calculi as a Late Complication of Fractures and Gunshot Wounds.—

- ⁸³ PAUL, *Canad. Med. Assoc. Jour.*, 1922, xli, 638.
⁸⁴ ROSENSTEIN, *Zeits. f. Urol. Chir.*, 1926-7, xxi, 326.
⁸⁵ ROSENSTEIN, *Med. Welt*, 1930, iv, 395.
⁸⁶ BLOCK, *Zeits. f. Urol.*, 1931, xxv, 332.
⁸⁷ MARSHALL, *U.S. Nav. Med. Bull.*, 1931, xxix, 472.
⁸⁸ BOSHAMER, *Arch. f. Orthop. u. Unfall-Chir.*, 1932, xxxii, 84.
⁸⁹ VOLKMANN, *Arch. f. klin. Chir.*, 1932, clxxi, 86.

Spontaneous Fractures in Paget's Disease.—

- ⁹⁰ ROBERTS and COHEN, *Proc. Roy. Soc. Med.*, 1926, xix, 14.

Albers-Schönberg's Disease.—

- ⁹¹ ALEXANDER, *Amer. Jour. Roentgenol.*, 1923, x, 280. (Review of literature to 1921.)
⁹² DAVIS, *Arch. of Surg.*, 1922, v, 449.
⁹³ CLAIRMONT and SCHINZ, *Arch. f. klin. Chir.*, 1924, cxxxii, 347.
⁹⁴ KRAUS and WALTER, *Med. Klin. Berl.*, 1925, xxi, 19.
⁹⁵ LAUTERBURG, *Schweiz. med. Woch.*, 1926, vii, 441.
⁹⁶ COHN. MAX, and SALINGER, *Med. Klin.*, 1927, xxiii, 825.
⁹⁷ BERNHARDT, *Klin. Woch.*, 1926, v, 415.
⁹⁸ BUSCHKE and OLLENDORFF, *Dermatol. Woch.*, 1928, lxxxvi, 257.
⁹⁹ D'ISTRIA, *Radiol. med.*, 1928, xv, 473.
¹⁰⁰ MERRILL, *Amer. Jour. Roentgenol.*, 1929, xxi, 361.
¹⁰¹ SEAR, *Brit. Jour. Surg.*, 1927, xiv, 657.
¹⁰² PRIRIE, *Amer. Jour. Roentgenol.*, 1930, xxiv, 147.

Kienböck's, Preiser's, Panner's Diseases, etc.—

- ¹⁰³ PREISER, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1910, xv, 189; xvi, 360.
¹⁰⁴ HAENISCH, *Ibid.*, xvi, 239.
¹⁰⁵ KIENBÖCK, *Ibid.*, 77.
¹⁰⁶ HIRSCH, *Ibid.*, 223.
¹⁰⁷ BUCKMAN, *Ann. of Surg.*, 1928, lxxxvii, 892.
¹⁰⁸ KÜHLER, *Münch. med. Woch.*, 1908, xlv, 1923.
¹⁰⁹ PANNER, *Acta Radiol.*, 1922.
¹¹⁰ FREIBERG, *Jour. Bone and Joint Surg.*, 1926, viii, 257.

Calcification of Supraspinatus Tendon.—

- ¹¹¹ PAINTER, *Boston Med. and Surg. Jour.*, 1907, clvi, 345.
¹¹² CODMAN, *Ibid.*, 1908, clix, 533, and 1911, clxv, 115.
¹¹³ BRICKNER, *Amer. Jour. Med. Sci.*, 1915, cxlix, 351.
¹¹⁴ ELMSLIE, *Brit. Med. Jour.*, 1931, ii, 898.
¹¹⁵ ELMSLIE, *Brit. Jour. Surg.*, 1932, xx, 190.
¹¹⁶ WATSON JONES, *Brit. Med. Jour.*, 1931, ii, 898.

Calcification in the Hæmangiomas.—

- ¹¹⁷ RUGGLES, *Amer. Atlas Stereoroentgenol.*, 1916, i, 82.
¹¹⁸ RUGGLES, *Amer. Jour. Roentgenol.*, 1919, vi, 512.
¹¹⁹ WAKELEY, *Arch. Radiol. and Electrol.*, 1921, xxv, 343.
¹²⁰ RAYOLD, *Jour. Radiol.*, 1924, iii, 231.
¹²¹ DUNN, *Ann. of Surg.*, 1925, lxxxii, 880.
¹²² JOHNSON, *Jour. Amer. Med. Assoc.*, 1928, xc, 1108.

Calcification in Meningeal Hæmangiomas.—

- ¹²³ KRABBE, *Rev. neurol.*, 1929, i, 1152.
¹²⁴ KRABBE and WISSING, *Acta radiol.*, 1929, x, 523.
¹²⁵ PÉPIN, DECHAUME, and BOUCOMONT, *Lyon méd.*, 1932, cxlix, 249.

Calcification in Raynaud's Disease.—

- ¹²⁶ THIBIERGE and WEISSENBACH, *Ann. de Dermatol. et Syph.*, 1911, ii, 129.
¹²⁷ FERNET and NAHAN, *Bull. Soc. de Dermatol. et Syph.*, 1919, xxvi, 266.
¹²⁸ WEIL, WEISSMANN-NETTER, *Ann. de Dermatol. et Syph.*, 1924, v, 724.
¹²⁹ LEHRNBECHER, *Beitr. z. klin. Chir.*, 1928, cxlii, 380.
¹³⁰ WEISSENBACH, VIGNAL, and GUILLAUMIN, *Bull. Soc. de Dermatol. et Syph.*, 1929, xxxvi, 910.
¹³¹ WEISSENBACH, TRUCHOT, FRANCON, and LAUDAT, *Bull. Soc. méd. Hôp. de Paris*, 1931, xlvii, 837.
¹³² GARCIN, BERTRAND, LAUDAT, and CACHIN, *Ibid.*, 1931, xlvii, 1036.

Calcification of Intervertebral Discs.—

- ¹³³ CALVÉ and GALLAND, *Jour. de Radiol. et d'Electrol.*, 1922, vi, 21.
¹³⁴ BARON, *Zentralb. f. Chir.*, 1924, li, 250.
¹³⁵ HORENSTEIN, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1927, xxxv, 713 (with literature).
¹³⁶ KRONENBERGER, *Röntgenpraxis*, 1929, i, 898.
¹³⁷ DITTRICH, *Amer. Jour. Surg.*, 1929, vii, 840.
¹³⁸ SCHMORL, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1929, xl, 18.
¹³⁹ BRETON, *Jour. de Radiol. et d'Electrol.*, 1929, xiii, 277.
¹⁴⁰ ISRAELSKI and POLLACK, *Beitr. z. klin. Chir.*, 1930, cl, 476.
¹⁴¹ LYON, *Arch. f. Orthop.*, 1930, xxviii, 717.
¹⁴² BARSONY and KOPPENSTEIN, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1930, xli, 211.
¹⁴³ ROVEKAMP, *Röntgenpraxis*, 1930, ii, 186.
¹⁴⁴ MANTOVANI, *Arch. franco-belges de Chir.*, 1930, xxvii, 488.
¹⁴⁵ ROEDERER, *Bull. et Mém. Soc. des Chir.*, 1931, xxiii, 596.
¹⁴⁶ CALVÉ and GALLAND, *Rev. d'Orthop.*, 1931, xviii, 782.
¹⁴⁷ RATHCKE, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1932, xlii, 66.

Calcification of Semilunar Cartilages.—

- ¹⁴⁸ MANDEL, *Arch. f. klin. Chir.*, 1927, cxlvi, 149.
¹⁴⁹ WERWATH, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1928, xxxvii, 169.
¹⁵⁰ BRUCHOLZ, *Röntgenpraxis*, 1929, i, 888.
¹⁵¹ SCHWARTZ, *Ibid.*, 1930, ii, 380.
¹⁵² ISRAELSKI and POLLACK, *Beitr. z. klin. Chir.*, 1930, cl, 476.
¹⁵³ ISRAELSKI, *Amer. Jour. Roentgenol.*, 1931, xxv, 85.
¹⁵⁴ HENRICHSSEN, *Röntgenpraxis*, 1932, iv, 403.
¹⁵⁵ MERCER, *Orthopaedic Surgery*, 1932, 426. London.

Subperiosteal Ossification.—

- ¹⁵⁶ WATSON JONES, *Brit. Med. Jour.*, 1930, ii, 592.
¹⁵⁷ WATSON JONES, *Canad. Med. Assoc. Jour.*, 1931, xxiv, 803.

Ossification of the Tendo Achillis.—

- ¹⁵⁸ SANTON and PERON, *Paris méd.*, 1922, xii, 342.
¹⁵⁹ STEIGER, *Wien. klin. Woch.*, 1925, xxxviii, 1012.
¹⁶⁰ HAGLUND, *Zeits. f. orthop. Chir.*, 1927, xlix, 49.
¹⁶¹ PATEL, *Lyon chir.*, 1931, xxviii, 351.
¹⁶² BRUMAGH, *Jour. Bone and Joint Surg.*, 1932, xiv, 933 (with bibliography).
¹⁶³ MALLINSON, *Brit. Med. Jour.*, 1932, ii, 836.
¹⁶⁴ WATSON JONES, *Ibid.*, 943.

A CASE OF HYPERPARATHYROIDISM WITH CERTAIN UNUSUAL FEATURES.

By T. W. MIMPRISS,

SURGICAL REGISTRAR, ST. THOMAS'S HOSPITAL,

AND R. W. BUTLER,

LATE CHIEF ASSISTANT, ORTHOPEDIC DEPARTMENT, ST. THOMAS'S HOSPITAL.

THE syndrome of hyperparathyroidism with a parathyroid tumour and changes in the bones has received so much attention in recent years that the main object in reporting the following case is to describe its occurrence in an adolescent patient, in whom growth is still taking place rapidly, and in whom therefore the bone changes are somewhat atypical.

CASE REPORT.

A stable boy, aged 17, was admitted to St. Thomas's Hospital on May 31, 1932, with a history of three months' pain in the knees, causing increasing difficulty in walking. Pressure over the long bones caused pain.

His appearance and mentality were those of a child of 14 and he had grown very little during the last three years.

Late rickets was suggested by a bossed forehead, enlargement of the costo-chondral junctions and of the radial, femoral, and tibial epiphyses, and muscular hypotonia was marked. Radiograms of the knees and wrists showed transverse bands of dense bone at the metaphyses, separated from the epiphysal lines by a narrow area of irregular ossification (*Fig. 341*). The bones generally showed some decalcification. In the shafts of the femora and one tibia were localized areas of rarefaction, suggesting early cyst formation.

The possibility of early renal rickets was not supported by an examination of the renal function.

Urine.—Specific gravity, 1022. No abnormal constituents found. Blood-urea : 34 mgrm. per 100 c.c.

Urea-concentration Test.—Fifteen grm. of urea were given. Percentage of urea in urine was : 1st hour, 2.1 ; 2nd hour, 2.2 ; 3rd hour, 2.4.

The blood-vessels were normal.

He had a small, dark-red tumour arising from the gum in front of the lower first bicuspid tooth on the right side. The teeth were normally erupted and an X-ray plate showed no change in the mandible. The serum calcium was raised to 14.6 mgrm. per 100 c.c., while the plasma phosphorus was 4.7 mgrm. per 100 c.c. His weight at this time (May 21) was 5 st. 12 lb. 10 oz.

He was kept under observation until July 31, with a tentative diagnosis of early hyperparathyroidism, his condition remaining unchanged.

At this time his serum calcium had risen to 18 mgrm. per 100 c.c. and his plasma phosphorus had fallen to 2.9 mgrm. per 100 c.c., while at the same time his renal function had deteriorated considerably. His urine contained a trace of albumin and numerous hyaline and epithelial casts. The

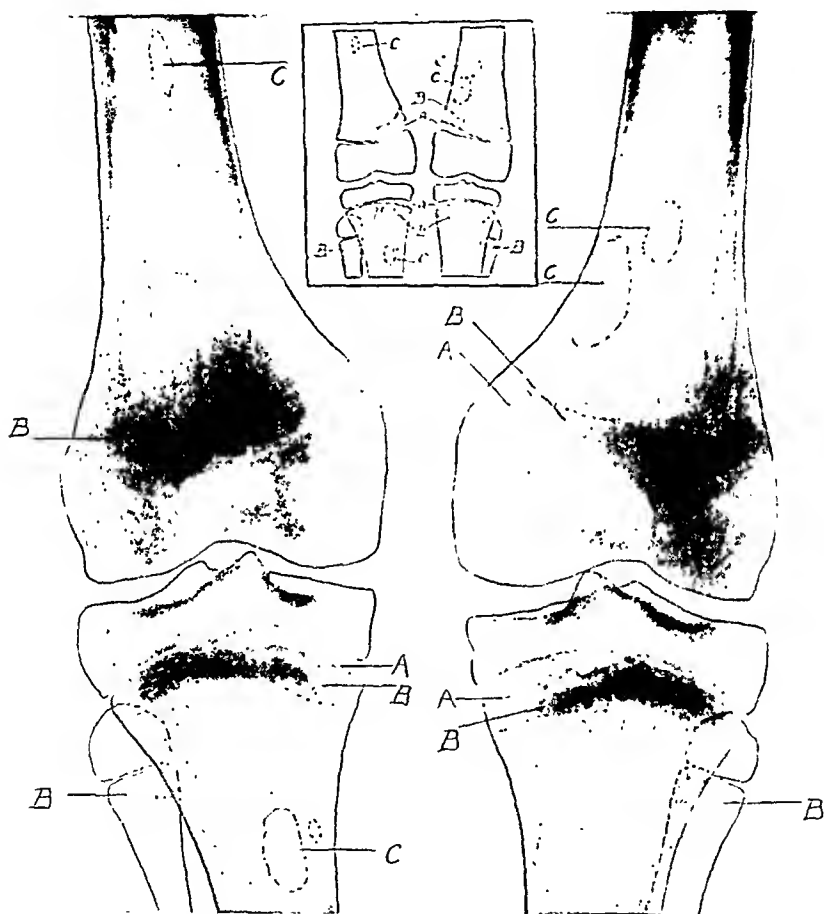


FIG. 341.—X-ray of knees before operation, taken in July, 1932. Showing: *A*, Irregular widening of epiphyseal lines; *B*, Transverse bands of increased density at the metaphyses; *C*, Localized areas of decreased density suggesting early cyst formation.

blood-urea was 62 mgrm. per 100 c.c. A urea-concentration test was as follows (15 mgrm. of urea given)—percentage of urea in urine: 1st hour, 1.9; 2nd hour, 1.9; 3rd hour, 1.4.

Frequent vomiting now became a notable feature, and by the end of July the patient weighed only 4 st. 12 lb., a stone less than when he was first seen at the end of May. The tumour of the jaw had increased

in size and a similar tumour appeared near the first lower molar on the left side.

Radiographic examination of the bones now showed that the irregular ossification at the metaphyses remained unaltered. The cystic areas were still present in the tibiae and femora, and others had appeared in the right radius and also in the mandible underlying the tumours visible in the mouth (*Fig. 342 A*). The cystic areas in the long bones were more definite than in the previous radiograms. A radiogram of the lumbar region showed no evidence of renal calculi.

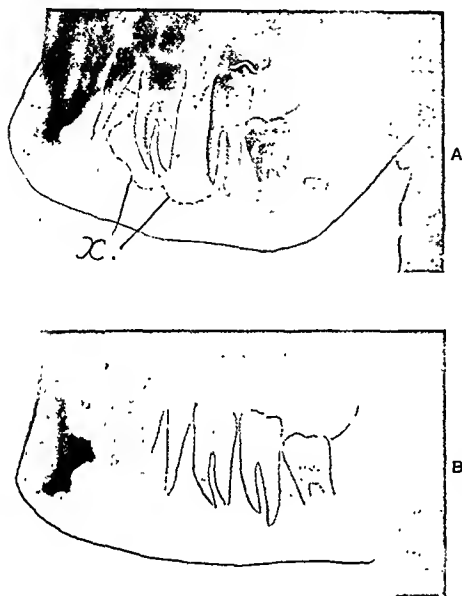


FIG. 342.—A, Mandible in July, 1932, before operation: *x* shows erosion of bone and separation of teeth at site of osteoclastoma. B, Mandible six months after operation. X-ray appearance is now normal.

yellow-brown tumour, 12 mm. in length, was found embedded in the anterior surface of the thymus. The tumour was excised with surrounding thymus tissue. At the same time one of the tumours of the jaw was partially removed for histological examination, and was found to be a typical osteoclastoma, containing numerous osteoclasts.

PATHOLOGICAL REPORT.—The retrosternal tumour (*Fig. 343*) was examined by Professor L. S. Dudgeon, who reported upon it as follows:—

The tumour is encapsulated; small blood-vessels are visible throughout, but the tissue is not very vascular. The tumour is composed almost entirely of one type of cell—the principal cell of the parathyroid gland. The nuclei are vesiculated to a varying degree, and contain nucleoli and a well-marked chromatin network. The cytoplasm is basophilic and shows granular marking. The cell membrane is distinct. The cells are densely packed throughout the tissue and are either polyhedral or arranged in columns about the blood-vessels. There are also a few small islets of cells surrounded by a thin fibrous capsule. These consist of foam cells of various sizes containing one or more

The patient was now very emaciated, vomiting frequently, and getting rapidly weaker. It was decided to explore the neck for a parathyroid tumour, although no tumour was palpable.

On Aug. 4, the day previous to operation, the serum calcium was 18.8 mgrm. per 100 c.c. and the plasma phosphorus was 2.9 mgrm. per 100 c.c., while a urea-concentration test gave the following reading—percentage of urea in urine: 1st hour, 0.8; 2nd hour, 0.9; 3rd hour, 0.8. The blood-urea was 66 mgrm. per 100 c.c.

OPERATION.—On Aug. 5 the neck was explored by Mr. W. H. C. Romanis. At the usual sites of occurrence of the parathyroid bodies no tumour was evident. A search was made in the retrosternal region and an ovoid

nuclei. The nuclei are relatively small and pale. The cytoplasm is pale and finely granular. No oxyphil cells were seen in the sections.

SUBSEQUENT PROGRESS.—The patient stood the operation well, and a striking improvement in his general condition followed rapidly. The frequent vomiting which had occurred before operation immediately ceased. His appetite, which had been small and capricious, improved steadily, while

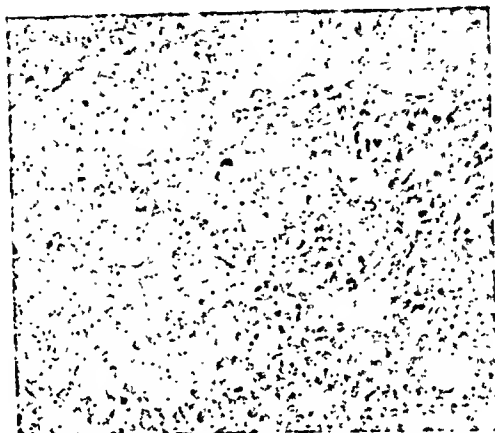


FIG. 343.—Microscopical appearance of tumour removed.

his weight, which was 4 st. 12 lb. 9 oz. on Aug. 4, the day before operation, was 5 st. 10 lb. 6 oz. on Aug. 18. and 6 st. on Aug. 22. The bone pains and bone tenderness were gone within a few days. The serum calcium and phosphorus readings underwent a sudden change following the operation.

Aug. 4.—The day before operation: Serum calcium 18.8 mgrm. per 100 c.c. Plasma phosphorus 2.9 mgrm. per 100 c.c. Blood-urea 66 mgrm. per 100 c.c.

Aug. 5.—Operation.

Aug. 6.—Serum calcium 12.7 mgrm. per 100 c.c. Plasma phosphorus 2.14 mgrm. per 100 c.c. Blood-urea 66 mgrm. per 100 c.c.

Aug. 8.—Serum calcium 8.8 mgrm. per 100 c.c. Plasma phosphorus 2.3 mgrm. per 100 c.c.

Aug. 12.—Serum calcium 10 mgrm. per 100 c.c. Plasma phosphorus 2.38 mgrm. per 100 c.c. Blood-urea 28 mgrm. per 100 c.c.

Aug. 18.—Serum calcium 10.9 mgrm. per 100 c.c. Plasma phosphorus 4.32 mgrm. per 100 c.c. These last readings are within normal limits.

Immediately after the operation the patient had been placed on a high calcium diet. It is interesting to note that there were no manifestations of tetany.

On Aug. 29 the patient was transferred to a convalescent home. He was then in good general health and putting on weight rapidly. The pain in his limbs had disappeared and he was able to walk.

On Oct. 10 the boy was seen again. He could walk easily and was in

good general health. His serum calcium was 10.8 mgrm. per 100 c.c. and his plasma phosphorus was 4.9 mgrm. per 100 c.c. The tumours of the jaw were slightly smaller. Radiograms of the bones showed no alteration from those taken before operation, the changes at the metaphyses and the cystic areas being still apparent. He was sent back to his home, with instructions to report again in the new year.

On March 6, 1933, the patient was again admitted to St. Thomas's Hospital for observation. He was then in good health, walking normally and anxious to resume work. His weight had increased to 7 st. 12 lb. Of

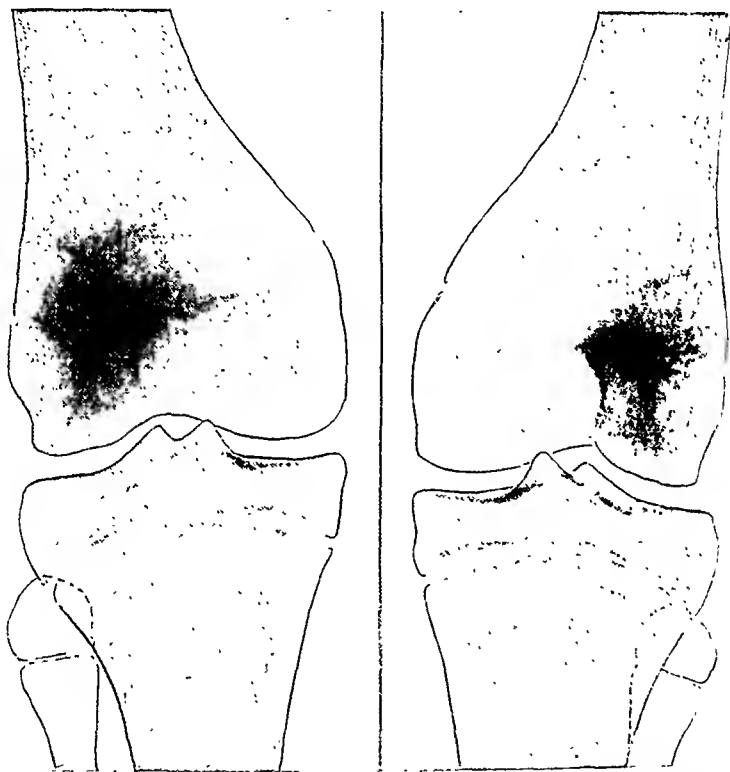


FIG. 344.—X-ray of knees six months after operation. The epiphysal lines are now regular. The bands of increased density at the metaphyses and the early cyst formation in the diaphyses have disappeared.

the two tumours of the mandible, one had completely disappeared, while the other was smaller in size. Radiographic examination of the bones showed that no new cyst formation had occurred, while the cysts previously reported were barely recognizable on the X-ray plate. Moreover, the metaphyses were now practically normal (*Figs. 342 B, 344*).

Biochemical tests also bore evidence of the return to normal metabolism. The serum calcium was 11 mgrm. per 100 c.c. and plasma phosphorus 5 mgrm. per 100 c.c. No abnormal constituents were present in the urine. His urea-concentration test was as follows—percentage of urea in urine: 1st hour.

3.4: 2nd hour. 3.5: 3rd hour. 3.9. Blood-urea 32 mgm. per 100 c.c. (Fig. 345).

No treatment was advised for the one remaining tumour of the mandible, since it was becoming smaller. This small tumour and the cystic areas still faintly visible in the long bones were at this time the only abnormal features still persisting.

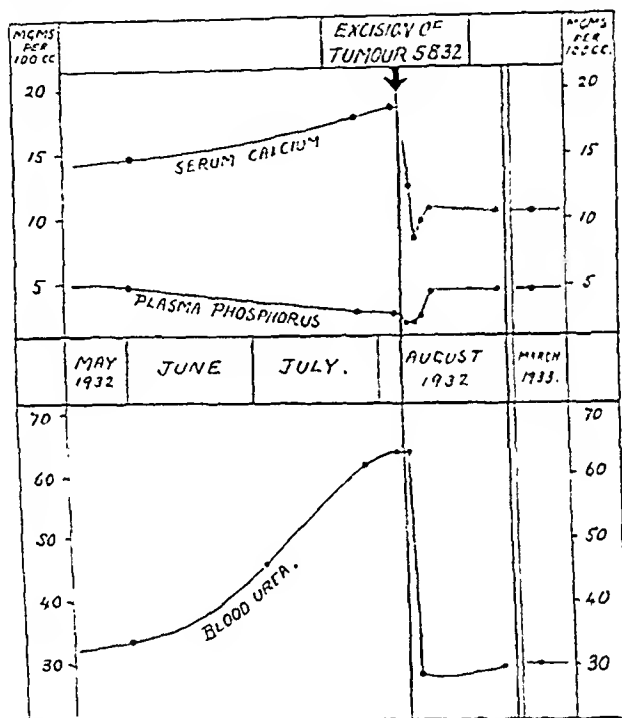


FIG. 345.—Table to show relationship of changes in serum calcium, plasma phosphorus, and blood-urea.

COMMENT.

This case is a well-marked example of hyperparathyroidism occurring at an unusually early age. The usual time of onset, according to Hunter,¹ is between 30 and 55.

The unusual radiographic appearance of a well-defined transverse band of increased density at the metaphyses as an early bone change is probably associated with the presence of active epiphysial growth. This metaphysial change does not appear to have been recorded as a manifestation of hyperparathyroidism with a parathyroid tumour, although Duken² in 1928 has reported exactly similar changes and published reproductions of the radiograms in the case of a girl aged 14, diagnosed as late rickets, who subsequently showed the changes of osteitis fibrosa. In his case the blood calcium showed a progressive rise and the plasma phosphorus was low, but at the

time of reporting the case no suggestion had apparently been made of the presence of a parathyroid tumour.

Another unusual feature of this case is the remarkable failure of renal function with the progress of the disease, and its equally remarkable recovery upon the removal of the tumour. This failure of renal function was not associated with the formation of renal calculi, which has frequently been recorded in hyperparathyroidism.

No exact explanation of this failure can be put forward, but it is of interest to note that Hunter and Aub³ have reported a marked rise of the non-protein nitrogen of the blood following the administration of large doses of parathormone in human beings.

An important feature of this case is the complete recovery of the patient after operation, in spite of his extremely precarious pre-operative state, and the absence of any permanent skeletal changes.

We are indebted to Professor Dudgeon for his kindness in furnishing the histological report, to Mr. W. Rowley Bristow and to Mr. W. H. C. Romanis for permission to publish this case, to Dr. Donald Hunter for his interest and advice in preparing this paper, and to Dr. Geoffrey Fildes for allowing us to reproduce his radiograms.

REFERENCES.

¹ HUNTER, D., *Brit. Jour. Surg.*, 1931-32, xix, 251.

² DUKEN, J., *Zeits. f. Kinderheilk.*, 1928, xlvj, 114

³ HUNTER and AUB, *Quart. Jour. Med.*, 1927, January.

THE OCCURRENCE OF SCROTAL HERNIA IN MICE UNDER TREATMENT WITH ŒSTRIN.

By HAROLD BURROWS.

EXPERIMENTAL PATHOLOGIST, RESEARCH INSTITUTE OF THE CANCER HOSPITAL (FREE),
LONDON.

DURING an experimental inquiry at this institute into the comparative effects of Œstrogenic and carcinogenic substances, scrotal hernias have been observed in some of the mice treated with Œstrin. The material used was pure keto-hydroxyŒstrin, for which we are indebted to the generosity of Dr. Girard.

Experiment 1.—Twenty-eight mice were treated twice weekly by the application of a solution (approximately 0·1 per cent in benzene) of Œstrin to the non-epilated skin of the interscapular region, by means of a small paint brush. In the course of this treatment a scrotal swelling had been observed in one of the mice by Mr. Woollard while performing his duties of laboratory assistant, and had been reported by him. When the experiment had lasted ten weeks three of these mice had scrotal hernias, bilateral in one and unilateral in the other two—one right and one left.



FIG. 346.—Two of the mice treated by subcutaneous injection of Œstrin and showing scrotal hernias are on the right, with two normal mice for comparison on the left.

Experiment 2.—During the same period eight male mice were treated with subcutaneous dosage of Œstrin in the right groin. During ten weeks five injections were given, each consisting of 0·5 c.c. of a 0·1 per cent solution of keto-hydroxyŒstrin in olive oil. At the end of this time five of the eight mice were found to have bilateral reducible scrotal hernias (Figs. 346-348).

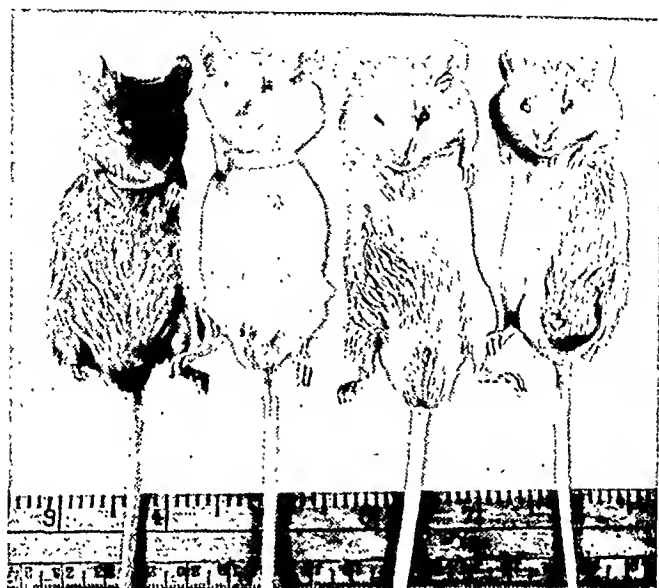


FIG. 347.—Two mice with hernias from the same series of œstrin-treated animals *vis* on the right, with two normal mice on the left. The mice shown in this and the preceding figure are under urothane anaesthesia.



FIG. 348.—Another of the same series of œstrin-treated mice. (This mouse is shown, with the hernia opened, in Fig. 350.)



FIG. 349.—Mouse treated with biweekly applications of œstrin (0.01 per cent) to the skin of the back and killed forty-seven days after the first application. Large serotal hernias are present.

Experiment 3.—Twenty mice were treated in the same way as those in the first experiment except that a more dilute solution of ketohydroxyoestrin was now employed (0.01 per cent in benzene). The experiment was commenced on Aug. 4, 1933. Thirty-five days later six of the thirteen mice still alive had double serotal hernias, and forty-seven days after the first application of oestrin eleven showed the presence of double serotal hernias (*Fig. 319*). One of the two mice remaining free from hernia was small and perhaps immature: it weighed only 15 gm. The other was of normal size (25 gm.) and apparently healthy.

Examination of 304 untreated male stock mice, and 276 male mice under treatment of various kinds not entailing the use of oestrin, failed to reveal a single serotal hernia, nor has this condition in mice been observed on any previous occasion by the author. The results are summarized in the table below.

INCIDENCE OF SCROTAL HERNIA IN MALE MICE UNDER TREATMENT WITH OESTRIN.

		METHOD OF APPLYING OESTRIN	PERIOD BETWEEN BEGINNING OF EXPERIMENT AND DATE OF OBSERVATION	NUMBER OF MICE SURVIVING AT THE DATE OF OBSERVATION	NUMBER OF HERNIAS
			Days		
Mice Treated with Oestrin—					
Experiment 1	Painted on skin	68	28	3	
Experiment 2	Subcutaneous injection ..	66	8	5	
Experiment 3	Painted on skin	47	13	11	
Total				49	19
Mice Not Treated with Oestrin—					
a.	Six-weeks-old untreated mice	47	0	
b.	Six-months-old untreated mice	43	0	
c.	Miscellaneous untreated stock mice	214	0	
d.	Mice under various forms of treatment other than by the administration of oestrin ..		276	0	
Total				580	0

Among the male mice painted with oestrin in the first experiment, the one with a left serotal hernia died, and the presence of coils of ileum in the scrotum was verified. For further investigation two of the animals treated by subcutaneous injection were killed. In one of these the left half of the scrotum contained the caecum, while the bladder lay in the right half. The other mouse was preserved as a museum specimen to illustrate the phenomenon. In this animal the caecum occupied the left half of the scrotum, and coils of small intestine distended the right half (*Fig. 350*). In all these three mice the gut lay in front of the testicles and epididymis, concealing these organs together with the genital omentum, which was small and contained but little fat, in contrast to that of a normal healthy mouse.

DISCUSSION.

A doubt might occur whether mere applications of œstrin in benzene to the non-epilated skin of the back could produce any recognizable effect. The same solution, however, applied twice a week in a similar way to ten female mice, not only kept them in a constant state of œstrus, as tested by the ordinary method of microscopical examination of vaginal smears, but led in every instance to the accumulation in the vagina of a mass of keratinized epithelium sufficiently large to distend the canal, while the uterine cornua became so enlarged as to be readily palpable in the living animal. Ultimately

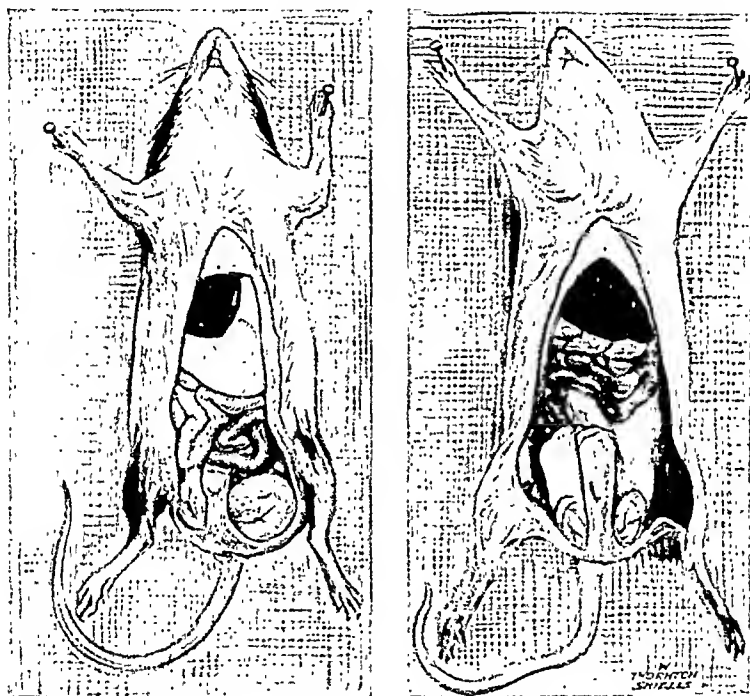


FIG. 350.—The mouse on the left had been treated by subcutaneous injections of œstrin. A double scrotal hernia is shown. The left hernia contains the cecum, and the right hernia is occupied by small intestine. A normal mouse is shown on the right for comparison. In both mice the scrotal coverings have been slit open through their whole extent, and a mesial strip of skin, including the penis, has been left *in situ*. The band of pale-coloured tissue which extends upward from each testis to the abdomen in the normal mouse is composed of fatty tissue and is spoken of in the text as the genital omentum.

the mice died from pyometra and perimetritis or from urinary obstruction with hydronephrosis. Such results indicate that ketohydroxyœstrin in the medium used is readily taken up through the healthy skin, although the possibility that some may have been inhaled as dust or ingested by the mice licking one another cannot be ignored.

In the mouse, as in the rat, the scrotum is a direct pouch of the peritoneum, and there is no greatly narrowed portion intervening which could well be described as a funicular canal, nor is there any difficulty in

manipulating the testicle into and out of the abdominal cavity. On dissection the aperture between the scrotal pocket and the peritoneal cavity seems so wide as to cause surprise that hernia in these animals does not often occur.

To the eye it may appear at first as though the testicle itself, together with the fatty genital omentum which is attached to the epididymis and spermatic cord and projects upward from this attachment into the abdomen, might perhaps by their bulk form an efficient barrier against the descent of intestine (*Fig. 350*). If this were the only protection against hernia, it would be removed by emaciation. These hernias have not been observed in emaciated mice: nor have they been seen as an accompaniment of ascites. Atrophy of the testicle is one of the consequences of administering œstrin to male mice, and it may be perhaps that the testicular atrophy is favourable to the development of hernia. But it can hardly be a cause, as the occurrence of hernia is an early phenomenon in the conditions now under discussion, and may be present before the diminution in size of the testicle has attained an advanced degree. Again, in rats, whose inguinal anatomy is comparable with that of the mouse, the author has seen extreme atrophy of the testicles without any sign of hernia.

Another point has to be considered. Among the several results of administering œstrin to male mice is an enlargement of the posterior lobes of the prostate with a consequent obstruction to the passage of urine, resulting in distension of the bladder with retention and overflow, and culminating in hydronephrosis.

These prostatic changes were first recorded in a recent paper by Lacassagne,¹ whose observations on the subject we were already prepared to confirm. It might be argued that the scrotal hernias were caused by the accentuated efforts necessary for micturition in these animals. Such a suggestion has little if any force as a complete explanation, seeing that hernia is a relatively early effect of administering œstrin, as shown by the table on p. 509, whereas symptoms referable to enlargement of the prostate and urinary obstruction have not been noted earlier than about the end of the fifth month after the first dose.

Attempts made on healthy mice to cause a protrusion of gut into the scrotum by pressure on the abdomen are unsuccessful. A similar failure attends the manœuvre when attempted on a normal mouse which has been recently killed, although the mesentery is long enough to allow excursion of the gut for the necessary extent. If putrefaction has become well established, on the other hand, coils of gut can be made occasionally to enter the scrotum by pressure on the abdomen.

In view of these facts one is led to believe that hernia is prevented in normal mice mainly or solely by the tonicity of the appropriate muscles, including the panniculus carnosus, which is strongly developed in these animals. The effect of œstrin might be to relax those layers of muscle which regulate the calibre of the passage-way between the abdominal cavity and the scrotum. The phenomenon may be cognate with the well-known fact that in some animals—the stag, for example—the testes descend into the scrotum during the rutting season only, remaining within the abdomen

during the rest of the year. On the other hand, Hisaw² found that the administration of an extract of sow's ovary to male pocket-gophers during the breeding season not only caused atrophy of the testicles but caused them to return into the abdomen. Some analogy perhaps may be observed between the production of hernia in male mice by the administration of œstrin and the relaxation of the muscular and ligamentous structures of the pelvis during the later stages of pregnancy when large quantities of œstrin are circulating in the body. In some species, e.g., the guinea-pig, the softening of the pelvic ligaments is of such a degree as to allow a wide separation of the two pubic bones during parturition. Courier³ and Brouha⁴ have shown that a similar relaxation of the pubic ligaments can be produced in the non-pregnant guinea-pig by the administration of œstrin.

The development of hernia in mice in the circumstances discussed above suggests interesting possibilities in connection with some cases of acquired hernia in man. With a further knowledge concerning hormones it is conceivable that we may be guided toward a method of preventing these lesions, and perhaps some others: abdominal proptosis, for example.

SUMMARY.

Nineteen cases of scrotal hernia were observed in 49 mice under treatment with œstrin.

Examination of 580 mice not treated with œstrin failed to reveal any instance of hernia.

I wish to express my indebtedness to the laboratory assistants both for their care of the animals and for their help in other technical matters.

REFERENCES.

¹ LACASSAGNE, A., *Comptes. rend. Soc. de Biol.*, 1933, cxiii, 590.

² HISAW, F. L., *Jour. Exper. Zool.*, 1925, xlii, 411.

³ COURRIER, R., *Proc. 2nd Internat. Congress Sex Research*, 1930, London.

⁴ BROUHA, L., *Comptes. rend. Soc. de Biol.*, 1933, cxiii, 406.

BENIGN GIANT-CELL TUMOUR OF THIRD CERVICAL VERTEBRA: A CASE REPORT.*

BY J. A. MACFARLANE AND ERIC A. LINELL, TORONTO.

A MAN (A. M.), aged 35, was admitted to the surgical wards of the Toronto General Hospital on Oct. 22, 1931, complaining of severe pain in the neck. On the previous evening he had been cranking the engine of his motor-car, when it backfired. He said that at that instant he was conscious of severe pain in his neck and shoulders. He made his way to his house with some difficulty. The pain continued and he passed a sleepless night.

Examination showed that he held his head and neck rigidly and that there was a moderate swelling in the mid-cervical region. Any attempt at movement caused severe pain. X-ray examination carried out shortly after his admission revealed a compression fracture of the body of the 3rd cervical vertebra, collapse apparently having taken place in the middle of the body, as there was very little evidence of change in its anterior aspect. The spaces between the 3rd and 2nd and the 3rd and 4th vertebrae were normal in width. No evidence of any disease could be seen in the adjacent vertebrae (*Fig. 351*).

From the history of the injury it seemed highly improbable that a simple traumatic compression fracture could have been produced in such a manner, and it was suspected that there must have been some pre-existing disease in the bone involved.

General physical examination was negative. No defect could be noted elsewhere in his osseous system. The function of his gastro-intestinal system was apparently normal and there was no history of loss of weight or failure of appetite. The thyroid was not enlarged. Rectal examination showed a normal prostate and there was no history of disturbance of urinary function.



FIG. 351.—X-ray of patient's cervical spine taken on admission to hospital, Oct. 22, 1931, showing fracture of body of 3rd cervical vertebra.

* From the Department of Surgery and the Division of Neuropathology, University of Toronto.

The man was treated by head extension on a Bradford frame, and as long as moderate extension was applied, he remained reasonably comfortable. At the end of ten days he developed a low-grade bronchopneumonia, from which he had recovered at the end of three weeks from the date of his admission. At this time the pain was considerably relieved and further investigations were carried out. A complete X-ray examination of the skeleton showed no sign of disease elsewhere. His urine was examined for blood and other abnormal findings, with negative results. X-ray examination showed the kidneys to be normal in size and position. Cystoscopy and pyelography were not carried out. Radiological investigation of the gastro-intestinal tract was undertaken and no evidence of new growth was found in stomach or colon. X-rays of the chest showed no evidence of intrapulmonary disease. A second plate of the cervical spine was made on Nov. 19 (*Fig. 352*), and this was



FIG. 352.—X-ray taken on Nov. 19, 1931. Shows extent of destructive changes in the body of the affected vertebra. There is evidence of angulation of the cervical spine.

compared with the previous plate and reported upon by the Radiological Department of the hospital as follows: "The upper part of the 3rd cervical vertebra has disappeared and there appears to be slightly more angulation at this examination than previously. No involvement of other vertebral bodies could be noted. Diagnosis at this time radiologically rests between primary or secondary new growth."

Consideration of the facts of the case up to this date seemed to favour the diagnosis of a giant-cell tumour. As has already been noted, there was neither clinical nor radiological evidence of primary new growth found in the gastro-intestinal tract, prostate, kidneys, or thyroid. There was no evidence clinically or radiologically that the man was suffering from osteomalacia, Paget's disease, or osteomyelitis. The possibility of a multiple myeloma with this lesion as the prim-

ary growth had to be considered, but one would have expected that evidences of similar disease elsewhere would be present at the end of four weeks. Chronic inflammatory disease of a tuberculous nature was also a possibility, but it has been pointed out that there was still no evidence of encroachment on the adjacent intervertebral discs. Syphilis was ruled out by a negative Wassermann reaction in blood and spinal fluid. Giant-cell tumour was, consequently, maintained as a working diagnosis, and it was thought unwise, on account of the inaccessibility of the lesion, to attempt the operative removal of tumour tissue merely for the purpose of confirming the diagnosis. The patient was therefore given deep X-ray therapy by the Radiological Department of the hospital. At the end of six weeks the upper part of his

trunk, the cervical spine, and the head were immobilized in a plaster jacket, and six weeks later he was allowed to go home. At this time he was reasonably comfortable except for pain which radiated to his shoulders if he remained erect for long periods. *Fig. 353* shows the X-ray appearances on Dec. 18, 1931.

He remained at home for six weeks, and then returned to hospital for the removal of his plaster. Inspection now revealed a shortening of his neck, giving him a 'bull-neck' appearance. The nasopharynx was increased in depth and he had a slight degree of voice change. X-rays at this time, Feb. 25, 1932 (*Fig. 354*), showed further collapse of the vertebra, but there was still no evidence of encroachment on the intervertebral discs or adjacent vertebrae. He was measured for a special celluloid supporting collar, with which he was allowed to go home on March 9.

Subsequently the patient was followed in the Out-patients' Department

and X-ray examination was carried out at monthly intervals. On each examination a little further destruction of the body of the vertebra was observed, until finally on June 22 (*Fig. 355*) only a small wisp of bone could be seen in the X-ray plate and still there was no evidence of encroachment on the adjacent structures. The man during this period was putting on weight, felt very well, and was anxious to go to work. He was allowed to return to work on June 27, and his company, rather than send him back to ordinary duties, allowed him to drive a small pony cart which was used for advertising purposes. When about to alight from the rear of this cart, the pony started suddenly and threw him off in such a manner that he struck his head violently on the pavement. He was taken in an unconscious



FIG. 353.—Further changes as seen on Dec. 18, 1931.



FIG. 354.—X-ray taken on Feb. 25, 1932. Shows well-marked angulation at site of diseased vertebra, also the well-marked backward convexity of the posterior pharyngeal wall.

condition to another hospital, where he was seen two hours later, obviously about to die. He was moribund and it was impossible to determine on physical examination whether he was dying from intracerebral injury entirely, or whether he had suffered a severe cord lesion from dislocation. He died a short time afterwards and permission to obtain the cervical spine was granted by the coroner's pathologist. Considerable difficulty was encountered in removing this without damage, and the fresh fracture, noted in the pathological report, at the end of the left lamina of the 3rd cervical vertebra may have been caused by this manipulation. There was gross evidence of a fractured skull with extensive intracranial hæmorrhage, and this would seem to have been the immediate cause of his death.

AUTOPSY.—The post-mortem examination revealed a fracture of the cranial base with laceration of brain tissue. The cervical spinal column was removed with its surrounding muscles.

The excised portion of the vertebral column was sawn sagittally, and examination of the cut surfaces revealed almost complete absorption of the body of the 3rd vertebra, there being only a small area of necrotic tissue intervening between the fibrocartilage at the distal end of the axis and that on the upper surface of the 4th cervical vertebral body (*Fig. 356 B*). The intervertebral disc of the distal surface of the axis had a convex inferior surface. The body of the axis formed, with the body of the 4th cervical vertebra, an angle of 145°



FIG. 355.—X-ray appearances as seen on June 22, 1932.

opening forwards. The backward dislocation of the axis had narrowed the antero-posterior diameter of the spinal canal to 1 cm. and had caused compression of the spinal cord. The dura mater ventral to the spinal cord at the level of maximum compression showed some thickening. There was no evidence of hæmorrhage into the membranes or into the subarachnoid space. Petechial hæmorrhages were visible in the spinal cord at its point of compression.

Examination of the external surface of the left half of the specimen showed a fresh fracture at the lateral end of the lamina of the 3rd cervical vertebra (*Fig. 356 A*). The lateral mass of this vertebra was practically completely destroyed, its place being taken by a necrotic cavity. No trace of the dorsal primary division of the left 3rd cervical nerve could be seen. On the right side a large area of the lateral mass of the 3rd cervical vertebra was replaced by soft necrotic tissue, and this tissue filled the space between the axis and 4th cervical vertebra. The 3rd cervical nerve was present and apparently normal on the right side.



FIG. 356.—A, Lateral view of left half of cervical spinal column showing: new fracture of lamina of 3rd cervical vertebra and absorption of the body of this vertebra with forward angulation of the column above this point. The tumour tissue was removed from the hole seen in the 3rd cervical vertebral body. No trace of the dorsal division of the 3rd cervical nerve could be found. B, Mesial surface of the right half of the cervical spinal column showing almost complete absorption of the body of the 3rd cervical vertebra with backward dislocation of the axis and severe compression of the spinal cord. Tumour tissue was found just above the upper border of the body of the 4th cervical vertebra.

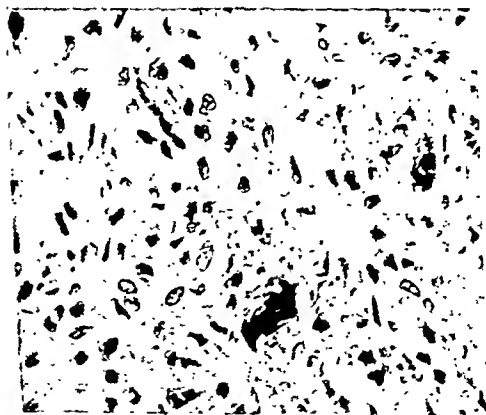


FIG. 357.—A typical low-power field taken to show a stroma of bipolar cells among which are seen three foreign-body giant cells typical of benign giant-cell tumour of bone. In the neighbourhood of the giant cell in the lower part of the field red blood-corpuscles can be seen lying free in the tumour tissue.

MICROSCOPICAL EXAMINATION.—Fragments of the necrotic tissue were scooped out from between the 2nd and 4th cervical vertebral bodies. Microscopically these were found to be tumour tissue, the matrix of which consisted of fusiform cells of which the nuclei were oval and, as a rule, showed a light chromatin content. Scattered through the tumour were large numbers of multinucleated foreign-body giant cells (*Fig. 357*). There were numbers of thin-walled blood-vessels throughout the tumour and a considerable amount of free hæmorrhage could be seen. A diagnosis of benign giant-cell tumour was made.

The dura mater ventral to the compressed spinal cord was thickened and contained a few collections of small round cells. There was no sign of infiltration of the membrane by tumour tissue.

Sections of the compressed spinal cord showed the two ventral horns of grey matter to be flattened and to lie in close apposition with each other. The nerve cells and the white matter of the cord stained satisfactorily. The blood-vessels were congested, but there was no evidence of hæmorrhage.

CONCLUSIONS.

The patient had a benign giant-cell tumour involving, and limited to, the body of his 3rd cervical vertebra. A pathological fracture of this bone occurred in October, 1931.

By a correlation of the clinical, radiographic, and post-mortem pathological findings, we are of the opinion that the backward dislocation of the axis and the compression of the cord increased gradually from the time of the pathological fracture to his death on June 27, 1932, and that his terminal injury was not responsible for the displacement of his cervical spine as revealed at the post-mortem examination.

The radiological investigation was performed by Dr. A. C. Singleton, of the Toronto General Hospital. We are indebted to Dr. George Laughlen, of the Toronto Eastern General Hospital, for his permission to examine the pathological material of the case and to publish this report.

ADENOPAPILLOMA OF THE STOMACH ASSOCIATED WITH LYMPHOID HYPERPLASIA OF THE DUODENUM.

BY A. E. WEBB-JOHNSON,

SURGEON TO THE MIDDLESEX HOSPITAL;

AND E. G. MUIR,

SURGICAL REGISTRAR, THE MIDDLESEX HOSPITAL.

BENIGN tumours of the stomach are still sufficiently rare to be regarded as clinical curiosities. Eliason and Wright¹ collected 560 cases from the literature and added 50 cases of their own, but the great majority of these were only found at autopsy. Eusterman and Senty,² in a review of cases at the Mayo Clinic, found that only 0.5 per cent of gastric neoplasms were benign. The published figures show that, of benign tumours of the stomach, myoma is found somewhat more frequently than papilloma. Associated lesions are present in many of the cases. Thus Balfour and Henderson³ found other lesions, such as ulcer or carcinoma, present in 22 out of 58 cases of benign gastric tumour.

The following case is of interest in that it was diagnosed before operation by the radiologist, Dr. Beath, of Belfast, and was associated with lymphoid hyperplasia of the first part of the duodenum, sufficiently marked to give rise to multiple small polypi.

CASE REPORT.

HISTORY.—The patient, a man of 72, complained of epigastric pain of eighteen months' duration. The pain was at first intermittent, but later became continuous and was made worse by food. Temporary relief was obtained by alkaline powders. The patient had suffered from constipation and believed that he had lost weight.

OPAQUE MEAL EXAMINATION (Dr. Beath).—

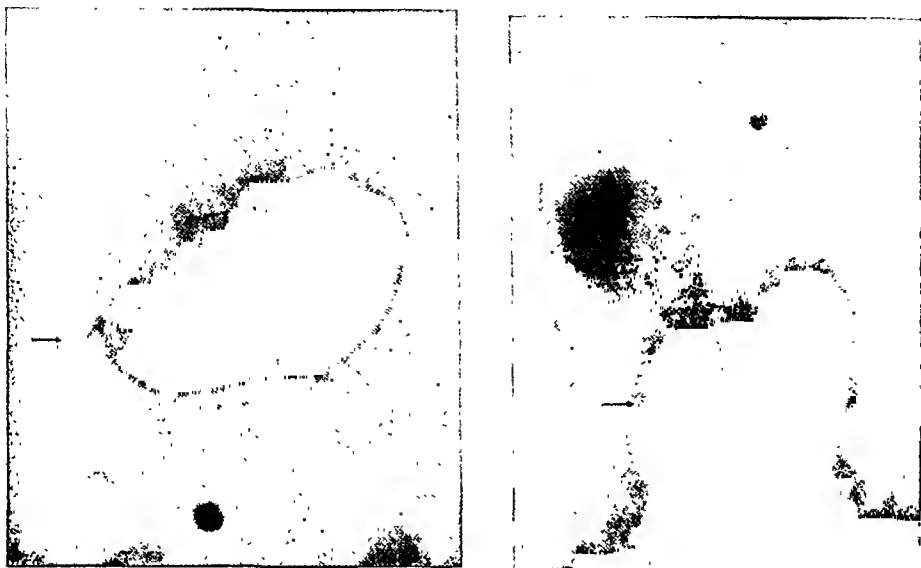
1. *Immediately After Meal (Erect).*—The stomach is normal in outline except at the extreme pyloric end, where there is a clear area, well defined and 'punched-out' looking. This is constant and can be seen on screening at all angles. Muscular tone is good and peristalsis normal. The duodenal cap fills readily and appears normal in outline. Initial emptying is at normal rate.

2. *Immediately After Meal (Prone).*—Confirms the above appearances in this position.

3. *Six Hours After Meal (Erect).*—The stomach is empty. There is a moderate degree of ileal stasis, with normal motility in the colon. The appendix is visible and looks normal.

4. *Twenty-seven Hours After Meal (Erect).*—There is considerable delay in the large intestine, which is spastic.

Findings.—There is a quite definite 'filling defect' (Figs. 358, 359) at the pyloric end of the stomach, but this does not present the usual appearances of a carcinoma. It is more suggestive of a papilloma growing from the



FIGS. 358, 359.—Barium meal showing 'filling defect' at pylorus.

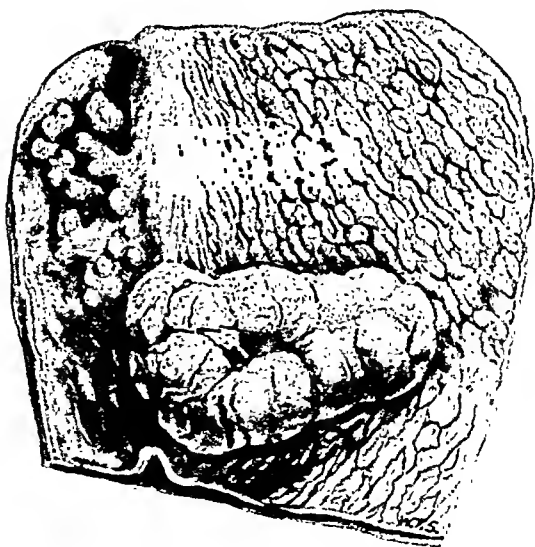


FIG. 360.—Resected portion of stomach showing adenopapilloma with hyperplastic lymph follicles of duodenum.

stomach wall. It would be impossible to discriminate definitely between the two possibilities, other than by a laparotomy.

OPERATION (May 10, 1932).—Laparotomy was performed and the stomach examined. A tumour could be felt at the pyloric end and partial gastrectomy was performed.

The patient made an uninterrupted recovery, and in February, 1933, reported that he was free from symptoms and was still gaining weight.

PATHOLOGICAL REPORT.—The specimen consists of $3\frac{1}{2}$ in. of stomach with a small piece of the duodenum (*Fig. 360*). Growing from the posterior wall of the stomach is an oval tumour, some 2 in. in length, with a short, broad base. There is no infiltration of the gastric wall nor any other lesion in the stomach. The duodenal mucosa on the posterior wall shows, just distal to the pylorus, a number of small polypoid processes, the largest being $\frac{1}{2}$ in. in diameter.

Histology.—Sections of the gastric tumour show an adenopapilloma with no evidence of malignancy. The small duodenal polypi consist of hyperplastic lymph follicles covered by mucosa.

COMMENTARY.

In the majority of cases the radiological diagnosis of such a condition as benign gastric tumour will be a tentative one, although in X-ray films we have examined of proved cases the appearances seem very similar and unlike carcinoma or ulcer. Moore,⁴ from a study of the radiological findings in 23 cases of benign tumour, concluded that these cases manifest certain signs which, if not definitely diagnostic, are at least suggestive. The stream of barium, as it enters the stomach, may be split in two by a tumour growing from the posterior wall. The filling defect is circumscribed and usually on the posterior wall. There is little or no disturbance of peristalsis except in those at or near the pylorus, nor is there any niche, incisura, or spasm.

In two cases described by Moore⁴ a "central, cyst-like filling defect was present in the duodenal cap". These were diagnosed as duodenal tumours, but at operation were found to be gastric growths which had prolapsed through the pylorus.

The pathological condition of the duodenum in this case is interesting, and in the reported cases of benign gastric tumour we do not find it mentioned as an associated lesion.

While there was no radiological evidence of prolapse in this case, the close proximity of the tumour to the pylorus suggests that intermittent prolapse, with irritation of the duodenal mucosa, may have been an etiological factor in the production of the lymphoid hyperplasia.

Partial gastrectomy would appear to be the most suitable method of treating benign gastric tumours. Balfour and Henderson³ described a series of cases in which the procedure used most frequently was transgastric excision through an incision in the anterior wall, and division of the pedicle by cautery, while partial gastrectomy was reserved for the larger tumours. They record one case in which carcinoma developed at the site of attachment of the pedicle.

Ewing⁵ believes that a number of cases of gastric carcinoma arise in adenomata. Since it is not possible to gauge the degree of malignancy of a tumour from a naked-eye examination, partial gastrectomy would appear to be the method of choice.

Our thanks are due to Dr. Beath, whose radiological report has been quoted verbatim, and to Professor James McIntosh for the use of pathological material.

REFERENCES.

- ¹ ELIASON, E. L., and WRIGHT, V. W. M., *Surg. Gynecol. and Obst.*, 1925, xli, 461.
- ² EUSTERMANN, G. B., and SENTRY, E. G., *Ibid.*, 1922, xxxiv, 5.
- ³ BALFOUR, D. C., and HENDERSON, E. F., *Ann. of Surg.*, 1927, lxxxv, 354.
- ⁴ MOORE, A. B., *Amer. Jour. Roentgenol.*, 1924, xi, 61.
- ⁵ EWING, J., *Neoplastic Diseases*, 671.

SPONTANEOUS DISLOCATION OF THE HIP IN CHILDHOOD.

BY P. N. RAY.

HON. SURGEON, CARMICHAEL MEDICAL COLLEGE HOSPITALS, AND ADDITIONAL SURGEON,
HOWRAH GENERAL HOSPITAL, CALCUTTA.

SPONTANEOUS dislocation may occur in any acute arthritis of the hip-joint as a complication. Cases¹ are recorded of dislocation in typhoid fever, rheumatism, scarlatina, and other exanthemata, as a result of acute non-suppurative infective arthritis. The following case is of interest on account of its occurrence during an attack of bacillary dysentery and its unusual termination.

CASE REPORT.

B. B., a Bengali female child, aged 6, was examined by me in consultation in December, 1931.

HISTORY.—The patient had a severe attack of bacillary dysentery in June, 1931. During convalescence several joints were affected by dysenteric synovitis. At first the left knee-joint became swollen, but within the next four or five days the right ankle, the right hip, and the left elbow were also involved. It took nearly six weeks before the effusion of the joints subsided, but the right hip-joint remained very painful and the patient was almost crippled and confined to her bed. In the other joints recovery was complete.

There was no history of any serious illness, or of any disability regarding the right hip-joint. She was a very healthy and bright child.

ON EXAMINATION.—The deformity of the right hip-joint was characterized by

flexion and adduction, but muscle spasm was not marked. The head of the femur was palpable over the gluteal region. There was marked limitation of movement in all directions, and all movements were painful. There was also pronounced wasting of the hip and thigh muscles. On measurement the shortening of the affected limb was found to be fully an inch. No abnormality could be detected in other joints. The patient was unable to stand or walk.



FIG. 361.—Before commencement of treatment: the acetabular fossa is occupied by an osteophytic mass.

The general health was very poor and she was reduced to a mere skeleton. The diagnosis of dorsal pathological dislocation was made. Reaction of degeneration, negative.

Skiagraphy.—The head of the femur presented no abnormality and there was practically no destruction of bone. The acetabular cavity was very shallow owing to the presence of an osteophytic mass (*Fig. 361*).

TREATMENT.—

1. *Local Treatment.*—The dislocation was reduced by the Lorenz method and kept immobilized in plaster-of-Paris. The patient was kept in bed for a fortnight and then she was encouraged to stand and walk with a high boot on the affected side. After eight weeks the casing was removed, and on examination the femoral head was found to lie in correct position and there was no tendency to recurrence of dislocation. A fresh plaster, however, was put on, but flexion was reduced to about 15° and abduction to 25° , and it was retained for eight weeks. The total period of fixation in plaster was four months. At the end of this period little difficulty was experienced in bringing the limb down to the normal position. The stability of reposition was maintained.

2. *General Treatment.*—The child was sent away to the country for a change of air, and other hygienic and dietetic measures were undertaken for the improvement of the general health.

FIG. 362.—Nine weeks after removal of plaster casing: showing the position of the femoral head and the shallowness of the acetabular fossa.

PROGRESS OF THE CASE.—After removal of the plaster casing, modified Thomas walking caliper splints were provided for six months for weight-bearing and walking on account of muscular wasting and weakness of limb. Massage and passive movements were continued throughout the period of treatment. At the end of one year muscular wasting was nearly made good.

During this prolonged period of treatment the affected hip-joint was examined under the X rays several times (*Fig. 362*). The latest skiagram (January, 1933) showed that the osteophytic outgrowth was so much reduced in size that the femoral head could be accommodated within the acetabular cavity (*Fig. 363*).



FIG. 363.—One year later: showing retrogression of the osteophytic outgrowth and lodgement of the femoral head within the acetabular fossa.

RESULT.—A mobile joint has been obtained and it is now completely free from pain. The final range of mobility is as follows: on flexion, the thigh forms an angle of about 20° with the abdomen; extension 25° beyond the long axis of the body; abduction 30° in the coronal plane; external rotation is slightly restricted; internal rotation and adduction are almost as good as on the healthy side. The limb can be easily placed in the 'tailor's position'. The patient walks without a limp.

COMMENTARY.

In attempting to arrive at a diagnosis of the condition, the possibilities which had to be considered were: (1) Congenital dislocation of the hip; (2) Infantile paralysis; and (3) Traumatic dislocation. The diagnosis was confirmed by the history of the case, skiagraphy, and electrical reactions of the wasted muscles. I have since seen one other case of spontaneous dislocation of the hip after non-suppurative typhoid arthritis in a girl of 3. The onset was insidious. The dislocation was reduced easily in this case. Dysenteric rheumatism or synovitis is said to be common in some epidemics, but the condition usually clears up without leaving any permanent deformity, though exceptionally permanent disability may result.²

Regarding the mechanism of the dislocation, emphasis has been laid on the over-distension of the joint cavity³ and the rupture of the capsule.¹ It is an interesting fact that in a case like the one described above, the

dislocation occurs very rapidly when there is marked flexor and adductor spasm. It has been pointed out that the pathological dislocation is caused by the derangement of the balanced action of muscles surrounding the hip-joint, as a result of muscle spasm and unequal distribution of muscle atrophy.⁴ Flexion, adduction, and internal rotation of the hip should therefore be considered a dangerous attitude. It is clear that the preventive treatment consists of early correction or prevention of this deformity with the restoration of muscular balance. Excessive or undue traction, on the other hand, may lead to the disruption of the softened ligaments and give rise to an obturator dislocation.⁵

The treatment of a patient after the deformity has been established is unsatisfactory, and frequently the luxation is a minor deformity compared to the associated deformities of other joints.⁶ Therefore early manipulative treatment is essential for satisfactory restoration of function. Too early interference, on the other hand, may flare up a quiescent infection, leading to more serious complications. Forceful manipulation may cause a fracture of the femur. With regard to complete restoration of function, prognosis is unsatisfactory. However early reduction is accomplished, some limitation of movement is to be expected and in many instances bony ankylosis or osteochondritis of the hip-joint. In later life osteo-arthritis may supervene because the changes initiated by such an infection of the joint may be continued by secondary degenerative alterations.⁷ In the case described above the incidence and retrogression of an osteophytic mass may be regarded as unusual. Generally the condition tends to be progressive.

SUMMARY.

1. A case of spontaneous dislocation of the hip following acute non-suppurative dysenteric synovitis has been described.

2. A painless mobile and stable joint has been obtained by manipulative reduction and immobilization in spite of the presence of a large osteophytic mass within the acetabular cavity.

3. After one year the osteophytic outgrowth has been so much reduced in size that the femoral head has been successfully accommodated inside the acetabular cavity.

I wish to thank Mr. Elmslie for his kind and helpful suggestions.

REFERENCES.

- ¹ WHITMAN, R., *A Treatise on Orthopaedic Surgery*, 1930, 9th ed., 420. London: Henry Kimpton.
- ² MANSON-BAHR, P., *Manson's Tropical Diseases*, 1929, 388. London: Cassell & Co.
- ³ CHOYCE, C. C., *System of Surgery*, 1925, iii, 982. London: Cassell & Co.
- ⁴ JONES, R. W., *Brit. Jour. Surg.*, 1926, July, 36.
- ⁵ ROMANIS, W. H. C., and MITCHNER, P. H., *The Science and Practice of Surgery*, 1930, 3rd ed., i, 606. London: J. & A. Churchill.
- ⁶ HART, V. L., *Arch. of Surg.*, 1928, Oct., 587.
- ⁷ ELSLIE, R. C., *Brit. Med. Jour.*, 1933, i, 1.

TWO CASES OF KELOID FORMATION, WITH COMMENTS.

By C. E. L. BURMAN, PIETERMARITZBURG.

AN unusual case of keloid reported by Professor Rutherford Morison¹ in 1920 is of special interest because it has been possible to follow developments for the last twelve years with the present condition of a case of recent date. (*Figs. 364, 365.*) The clinical characters are so similar that a possible theory as to the causation of the keloid condition, in the light of recent knowledge, is suggested.



FIG. 364.—Rutherford Morison's case, 1933.



FIG. 365.—Present case.

Professor Rutherford Morison said: "Keloids are interesting growths and require further research work to be expended upon them, because they form a link between the infectious and malignant growths. They possess a local malignancy so pronounced as to make it almost certain that in this case there will be recurrence of the growth which has been for the time so successfully removed. We also know that they are associated in some way with

sepsis and tubercle of chronic type; that on the skin, burn scars, which take a long time to heal, and scars resulting from tubercle, are their sites; and they are more common in the black than in the white races."

In an article "Observations and Reflections on Cancer in General Practice", utilizing this case, I stated:² "Clinically, he undoubtedly possessed that 'something' so essential to the manufacture of good, sound fibrous tissue, and which, at present, is the secret of nature in deciding the degree of malignancy of a growth. The development of good, sound fibrous tissue is always absent in the cancers which we recognize as extremely malignant, and recurrence is rapid. On the other hand, where the patient possesses the biochemical properties of producing good, sound fibrous tissue, the growth is always slow growing and of the scirrhus type. I am of opinion that it would be impossible to produce an external cancer in this native, so good are his fibrous forming properties. Chronic irritation in him produces no cancer cells, but active proliferating connective tissue cells, and yet removal is followed by local recurrence."

The present case of keloid, seen on Feb. 12, 1932, gave the following history:—

Well-built native, aged 30 years. Vaccinated at the age of 10 years. His ears, as is the native custom about puberty, were perforated so that ornaments could be inserted or carried in the openings. Shortly afterwards the growth of the lobe of the right ear started and slowly enlarged. In 1925 the growth, then about the size of a pigeon's egg, was removed, and owing to its return and increase of size it was again removed in 1929.

He consulted me with a view to further removal on account of severe itching and increase of the size since removal in 1925. Keloid growths were evident on the lobe of the right ear, under the chin, forehead, chest, on both arms in the region of the elbows, and on other parts of the body.

In 1926 he fell off his bicycle and scraped both arms. In 1929 he was attacked by other natives, and wounds (which were stitched) were received on the forehead and chest. In April, 1931, he was injured on the back of the head, and in November of the same year on the forehead.

On the back there were six scarifications which he says were done during infancy but showed no signs of keloid formation, and a thin papery scar on the outer side of the right leg, the result of a tear from barbed wire before he reached puberty.

In both this case and that reported by Rutherford Morison similar manifestations are evident: (1) Vaccination marks and scarifications produced before puberty show no keloid formation unless as rare exceptions; (2) Marked distension of the veins of the extremities; (3) Local recurrence after removal, with increase of size of growths; (4) Slow progress of growths where removal has not been carried out; (5) Injuries after puberty are followed by keloid growths; (6) No organic disease is evident other than the growths; (7) The enlargement of the penis; (8) Sepsis is present in the growths where surgical removal has been undertaken, and in Morison's case is slowly producing wasting, malnutrition, and a miserable existence.

Keloid formation is undoubtedly more common in natives and those with pigmented skins. The development of the medullary portion of the adrenal glands with the sympathetic system and their association with the connective-tissue cells of the skin, and more especially the layer of the stratum granulosum containing eleidin, the forerunner of the keratin of the stratum lucidum, may have some bearing in bringing about, at puberty, over-stimulation of these cells, with consequent overgrowth when a wound is produced and healing is necessary.

In Addison's disease we have disease of the medullary portion, with consequent loss of stimulation, but pigmentation. Similar analogies can be noted in disease of the thyroid, parathyroid, pituitary, and other glands with an internal secretion controlled by the sympathetic.

My thanks are due to Professor Rutherford Morison for permission to utilize his case; to Dr. Pinniger, of Ladysmith, Natal, for referring the present case; and to Sergt. Andersen, of the C.I.D. of Maritzburg, for the reproduction of the photographs.

BIBLIOGRAPHY.

- ¹ MORISON, R., *Brit. Jour. Surg.*, 1920, vii, 538.
- ² BURMAN, *Med. Assoc. S. Africa*, June 13, 1931.
- SCHAFER, *Essentials of Histology*, 1929, 12th edition.
- FRASER, *Manual of Embryology*, 1931.
- EWING, *Neoplastic Diseases*, 1928.

ACUTE INFECTIVE OSTEOMYELITIS OF THE TIBIA: USE OF WINNETT ORR TECHNIQUE.

By M. G. O'MALLEY,

PROFESSOR OF SURGERY, UNIVERSITY COLLEGE, GALWAY.

THE value of the Winnett Orr method in the treatment of compound fractures and chronic osteomyelitis is generally recognized; but apparently its application in acute osteomyelitis has not found favour in this country if one is to judge from recent text-books and publications.

At the Central Hospital, Galway, during the years 1930-32, 17 cases of acute infective myelitis of the tibia, out of a total admission of 31, were treated by the Winnett Orr method. Some of the cases were of a very severe type, two being complicated by septic arthritis of the ankle-joint.

There were 4 deaths in the whole series of 31 cases. Three deaths occurred in the 14 treated by open drainage, and there was one death in the 17 treated by the Winnett Orr technique. These figures are not given to prove the life-saving value of the latter method. Of the 3 fatal cases treated by open drainage, one, a boy aged 4½ years, died on the day following admission; another, a boy aged 10, died on the second day; and the third, a boy aged 8, died on the sixth day after operation. Two of the cases were moribund on admission, the other case might have been saved by an amputation, for which the consent of the relatives could not be obtained. The fatality among those treated by the Winnett Orr method was a boy aged 9 years, who died on the seventh day following operation. An amputation would have been more appropriate in this case.

A review of the 16 successful cases treated according to the Orr technique showed that an average of three dressings were necessary. The first dressing was usually left untouched for eight weeks. The subsequent dressings became rather intolerable in four weeks, and in many cases even earlier. The stench is never so pronounced in the acute as in the chronic cases.

Patients who come from good homes are treated as out-patients at the end of a week. The majority, however, come from poor out-lying parts of the county, and we find it necessary to detain them in hospital until healing is complete. The rural slums on the western seaboard provide us with many victims where home treatment is impossible.

Brief details of four of the more serious cases are given below:—

Case 1.—M. L., female aged 13 years, admitted to hospital on June 14, 1931. Acute osteomyelitis of the left tibia with septic arthritis of the ankle. Temperature 102°, pulse 124. Mildly delirious.

The lower three-fourths of the tibia were opened up and incisions were made into the ankle-joint. The wounds were packed with vaseline gauze and plaster-of-Paris was applied. Temperature and pulse were normal on the third day, and there was no pain after the second day. This case required redressing on the sixteenth day owing to the return of pain and temperature. Convalescence was smooth after the second dressing. Four dressings were required in all. The wounds were finally healed on Nov. 7. The result was excellent, with good function in the ankle-joint.

Case 2.—W. N., female aged 11 years, admitted on Nov. 17, 1931. Temperature 104° , pulse 120. Extensive involvement of the left tibia and ankle-joint. The whole of the shaft of the tibia was opened up and several incisions were made into the ankle-joint. Temperature and pulse returned to normal on the third day and the subsequent course was uneventful. Three dressings were required. The wounds finally healed on Feb. 21, 1932. This was a particularly gratifying result.

Case 3.—J. O. T., male aged 5 years, admitted on Dec. 9, 1931. Temperature 102° , pulse 120. Involvement of the whole of the tibial shaft with an abscess pointing in the popliteal space. An incision was made in the popliteal space and the abscess traced down along the inner side of the knee to the upper end of the shaft of the tibia. The shaft of the tibia was opened up in its whole length. The wound in the soft tissues extended from the popliteal space to the ankle. Temperature and pulse returned to normal after the fifth day. The wound finally healed on June 2, 1932. This case required five dressings.

Case 4.—M. R., male aged 7 years, admitted on Feb. 5, 1931. Temperature 103° , pulse 140; very ill. The whole of the shaft of the tibia was involved. Winnett Orr technique. Temperature and pulse normal on the sixth day. The wound finally healed on July 14. Four dressings were required.

Every case of the 16 under review required extensive exposure of the medulla. I have rarely seen a case here where simple incision into the periosteum or limited trephining of the metaphysis would suffice. I can only recollect two cases in the last five years. As already stated, the patients come from poor and remote districts where the services of a medical man are not readily obtained.

A review of the 11 successful cases treated by open drainage shows that convalescence is not at all smooth in a large proportion. A fluctuating temperature for some weeks occurred in 7 cases, in 2 it was associated with pyæmic abscesses. The charts show abundant evidence of the use of tonics and sera.

I have very little experience of the application of the Winnett Orr technique to acute infective osteomyelitis of other regions of the body. The method requires for its success a very good exposure of the infected area, with complete saucerization of the wound. It is easy to obtain such conditions in the tibia and perhaps the fibula and metatarsal bones. My colleague, Mr. MacDermott, has had very good results with the method in acute disease of the femur.

Seventeen cases would perhaps be considered a small number on which to base important conclusions; but all the patients had rather extensive disease of the medulla, and four were very severe infections. I think that a review of the cases proves that the method possesses the following advantages: (1) Economy of dressings; (2) Absence of pain, and ease in nursing; (3) The prevention of deformities; (4) A smooth convalescence with a lessening of the incidence of pyæmia. These are very solid advantages.

The one disadvantage is the smell. This is not at all bad with the first dressing. Good ventilation around the limb is the best way of avoiding it. There is in the hospital at present a patient who was admitted eight weeks ago with chronic osteomyelitis of the right tibia and left ulna. I treated both lesions by the Winnett Orr method. It has been necessary to renew the leg dressing once, but the upper limb dressing has given no trouble. I attribute this to the fact that the arm is kept outside the bedclothes and consequently gets good ventilation.

SHORT NOTES OF RARE OR OBSCURE CASES

LARGE CALCIFIED HYDATID CYST OF THE OMENTUM IMPACTED IN THE PELVIS AND CAUSING OBSTRUCTION OF AND HÆMORRHAGE INTO THE COLON.

BY H. WALDEN FITZGERALD, DUNEDIN, N.Z.

THE patient, a male of 70 years, was referred to me with the following history: Colicky pains in the abdomen, flatulence, constipation, 'tarry' and blood-stained stool, attacks of vomiting, and shortness of breath, for the last six months. The attacks of vomiting, colic, and constipation have become progressively and rapidly worse. There have been no urinary symptoms.



FIG. 366.—X-ray appearance of the condition.

The only incident in the previous history was, on the patient's story, a 'strained heart' thirty years ago while mountaineering, but at present his effort response is remarkably good.

ON EXAMINATION.—The respiratory and genito-urinary systems showed nothing of note. Cardio-vascular system: Few extrasystoles, blood-pressure 160/190, and otherwise fairly satisfactory. The central nervous system elicited no abnormality. Alimentary system: The abdomen was distended and definitely rigid and tender in the hypogastrium. No peristalsis was observed.

Per rectum the examining finger came immediately on to a large stony-hard immovable mass pressing directly on to the rectum from above and in front; the prostate was soft and uniformly slightly enlarged.

X-ray Examination (Dr. M. Barclay).—"Barium enema (July 26, 1932): The colon fills, revealing ballooning of the upper rectum, no tenderness, foreign body seen—(?) calcified mass. A large heavily calcified mass, measuring about

3 × 3½ in., lies centrally in the true pelvis, reaching from the pubic symphysis to the level of the pelvic brim. The barium enema was not obstructed, but the proximal portion of the rectum is narrowed, presumably owing to pressure. Findings are not suggestive of an intrinsic rectal lesion. It is impossible to see the lower sacrum and coccyx, which are overshadowed by the calcified mass. Elsewhere the bone is normal. Further investigation reveals the mass lying in the true pelvis, well in front of the anterior surface of the sacrum. The X-ray findings indicate that it is lying in the soft tissues between the rectum and the bladder." (Fig. 366.)

The Casoni Test (for hydatids).—Positive.

ON ADMISSION.—The patient was admitted to a nursing home on Aug. 4 after a severe collapse at his home following almost absolute constipation. He was very pale and his pulse rapid and rather poor. Repeated enemata gave huge results of 'tarry' and blood-stained, foul-smelling faecal material. Following these, and the administration of large quantities of fluids, digitalis, and rest in bed his condition improved markedly and the abdominal distension subsided to some extent. On admission there was copious vomiting of almost feculent vomit; this stopped following the repeated enemata.

PRE-OPERATIVE DIAGNOSIS.—(1) Calcified hydatid cyst of the omentum; (2) Dermoid cyst, calcified.

OPERATION (July 9).—Atropine and sodium amytal premedication. Ethyl chloride, and oxygen and ether anaesthesia. An attempt to catheterize the bladder failed owing to the mass pressing on the prostatic urethra. Medium Trendelenburg position. The abdomen was opened through a median supra-pubic incision and the small gut packed off. The omentum was adherent in the pelvis to the bladder and parietal peritoneum; these adhesions were separated. The calcified cyst, almost entirely filling the pelvis, and grooved antero-inferiorly by the prostate, was delivered without much difficulty. It was found to be growing within the layers of the omentum; the omentum was clamped and the mass removed. The omental stump was ligated in bundles. The whole of the colon was filled and distended with hard fecal masses. Rectum free from any growth and the stomach and duodenum normal. The cyst was not adherent at all in the pelvis, and had evidently grown in the omentum, possibly originating primarily from the liver, gravitating gradually to the pelvis, and there causing pressure symptoms. The abdomen was closed in layers without drainage. There was an uneventful convalescence except for a small abscess in the arm at the site of a hypodermic injection—this was opened and drained and healed without trouble.

POST-OPERATIVE DIAGNOSIS.—Calcified cyst of the omentum impacted in the pelvis causing pressure symptoms, the cyst being hydatid in origin.

PATHOLOGICAL REPORT (Otago University Pathology Department).—"The specimen consists of a calcified hydatid cyst 1½ × 3 × 3½ in. with a well-marked groove on the anterior aspect where it had surrounded the prostate. The weight is 9 oz. The calcified wall is 1½ in. in thickness and the contents are portions of ectocyst apparently belonging to daughter cysts and a quantity of amorphous material in which scolices can be found."

SUBSEQUENT PROGRESS.—There have been no untoward symptoms, and on examination at the end of January, 1933, everything was entirely satisfactory.

A CASE OF NEUROCYTOMA OF THE PELVIC SYMPATHETIC TRUNK.*

BY A. W. FARMER AND ERIC A. LINELL, TORONTO.

THIS case is reported because of the rarity of the condition and the unusual interest of the microscopical findings.

The patient, a boy of $4\frac{1}{2}$ years, came to hospital with the complaint of a sudden onset of urinary retention. Ribbon-like stools had been noticed for some weeks. The child was well nourished and did not appear acutely ill. Upon examination a hard mass was felt in the left lower quadrant of the abdomen, extending beyond the mid-line, and rising about an inch above the



FIG. 367.—X-ray showing deformity and separation of segments of sacrum.

brim of the pelvis. The examining finger could be forced into the rectum with difficulty. The mass practically filled the pelvis and pushed the rectum forward and to the right side. The tumour was firm and smooth and was fixed to the sacrum.

An X-ray plate of the pelvis and sacrum showed considerable deformity and separation of the sacral segments (*Fig. 367*).

* From the Department of Surgery, Hospital for Sick Children, Toronto, and Division of Neuropathology, University of Toronto.

A biopsy was performed by excision of tissue from the posterior surface of the mass after removal of the coccyx. Later the tumour was excised in two stages. About one-third of the mass was removed through an abdominal incision and the remainder was resected four weeks later by an approach from below through the pelvic floor. The rectum was displaced forward against the symphysis pubis, the bladder being pushed up out of the pelvis. There was scarcely room for one finger to be inserted between the mass and the symphysis. The tumour was very easily separated from all surrounding structures except the sacrum, where it entered the anterior sacral foramina, and between the sacral segments.

The growth was considered to have invaded the foramina rather than to be growing out of them.



FIG. 368.—Low-power magnification of neurocytoma. Shows a number of multipolar nerve-cells lying in a stroma largely composed of fibroblasts. (Cresyl violet.)



FIG. 369.—High-power microphotograph. Shows peripheral ring of Nissl granules in cytoplasm of nerve-cells. The fibroblast nuclei are somewhat irregular in shape and size. Neurilemmal sheath-cell nuclei are distinguished by their round or short-oval shape. (Cresyl violet.)

A retention catheter was inserted before operation. Following operation this was removed, but the bladder became distended almost to the umbilicus and then there was a constant overflow of urine. Neither before nor after the operation was any disturbance of perineal sensation noted. According to word received from the family physician two months after the discharge of the patient from hospital, the ability to empty the bladder voluntarily has not yet returned.

As it was impossible to be sure of the complete removal of the growth from between the sacral segments, the region was subjected to deep X-ray therapy.

The mass was not removed in one piece, so that an accurate measurement of its size was impossible. It was roughly oval in shape, being 4 in.

in its long diameter, and about $2\frac{1}{2}$ in. in its short diameter. It was bluish-white in colour and uniformly firm in consistency. It showed in places nodular excreescences, which had a faintly yellow colour. On one surface of the tumour there was evidence that the growth had a thin fibrous capsule.

MICROSCOPICAL FINDINGS.—The tumour is composed mainly of fibrous tissue, scattered through which are bundles of non-medullated nerve-fibres and numbers of large multipolar nerve-cells. *Fig. 368* shows a low-power microphotograph of the tumour tissue stained with cresyl violet. Under high magnification (*Fig. 369*) the large multipolar nerve-cells of the tumour are seen to have a ring of well-defined, peripherally-placed Nissl granules. The fibroblast nuclei are of a long oval shape, showing some irregularity in their contour. Processes can be seen arising from the poles of these nuclei. In this microphotograph can also be seen a few neurilenal sheath-cell nuclei, distinguished by their round or short-oval shape.

Occasionally a nerve-cell can be seen undergoing division, and here and there are small collections of lymphocytes lying free in the tumour tissue. With Cajal's gold sublimate stain it is evident that there are large numbers of bundles of non-medullated nerve-fibres running through the tumour and that the main tumour mass is an intimate mixture of neoplastic non-medullated nerve-fibres and fibrous tissue. A silver impregnation picks out differentially the sheath-cell and fibroblast nuclei. With ammoniacal silver oxide the fibrous tissue bundles do not impregnate deeply, suggesting that the fibroblasts are somewhat immature. With Weigert's stain small medullated nerve-fibres can be seen running through the tumour in all directions.

Our thanks are due to Dr. I. H. Erb, Pathologist to the Hospital for Sick Children, Toronto, for his help.

CYST OF THE COMMON BILE-DUCT.

BY D. G. DUFF,

SURGEON, DENBIGHSHIRE INFIRMARY.

COMMON-DUCT cyst is infrequent enough to warrant report of a case showing unusual features. Less than one hundred cases have been published, and it is stated by Wilson¹ that only three cases have been diagnosed before operation.

HISTORY.—In February, 1933, I saw with Dr. Leiper, Llanfair Talhaiarn, a multipara 62 years old who had been bed-ridden for over three months suffering from recurring bouts of abdominal pain with vomiting and constipation. Notes from her National Health Insurance record card show that, previously healthy, she was first incapacitated seven years before, when she had "gastritis". In August, 1928, and October, 1932, she again had trouble, and thereafter symptoms became steadily worse. Her doctor noticed an abdominal swelling and sent her to a hospital in November, 1932. "Pneumonia" supervened and she was kept in bed for three months, but on return home the swelling was again noticed.

ON EXAMINATION.—I could find no more than an abdomen tender all over and peculiarly doughy to palpation, but on admission to Denbigh Infirmary a month later she had a large cystic immovable swelling in the upper abdomen as big as a football, and separate from the liver, which was pushed up about two inches. Bismuth enema showed a normal colon, and perabrodil injection gave a good picture showing normal renal pelvic anatomy and function. Other clinical tests revealed no abnormality, and a diagnosis of retroperitoneal cyst, possible cyst of the common bile-duct, was made.

OPERATION.—Operation disclosed an abdomen full of adhesions and with lymphatic channels throughout the peritoneal cavity full of grey-black pigment. A cystic swelling with tense grey walls occupied the position and had the relations of the lesser sac. The posterior wall of the stomach was so firmly bound down to its upper anterior surface that dissection was quite impossible. Aspiration gave dark bile under such tension that five pints were run off. The total contents were estimated to be eight pints. An opening was made and clots containing bile pigment and sediment were removed. A passage admitting the tip of the little finger was discovered flattened out in the posterior cyst wall and apparently leading to the ampulla of Vater. No other openings corresponding to the entrance of common bile, cystic, or hepatic ducts were found, but owing to the patient's condition little time was available for the search. It was impossible to explore the bile-passages outside the cyst, but by working through adhesions the gall-bladder and adjoining liver surface were exposed, both apparently perfectly normal. On the front wall of the cyst was a scar where, it was surmised, there had been a rupture some months before which had caused bleeding into the peritoneum and the adhesions mentioned. Hæmorrhage has been a frequent cause of death in these cases.

An anastomosis was made between the cyst and the highest available part of the gut, the jejunum 3 in. from its commencement. No clamps were used, and fine stitching was necessary owing to the vascular cyst wall. An opening to admit two fingers easily was made so that there would be free exit for any intestinal contents which might enter the cyst.

The patient made uninterruptedly good progress and is now very fit.

COMMENTARY.

Of interest is the unusual age of the patient, the average age at operation being 18,¹ and the fact that her symptoms developed so rapidly so late in life. The disappearance of the tumour made diagnosis at times quite impossible.

It may be noted incidentally that though infection in these cysts is frequent and biliary stasis necessarily present, true gall-stones have not been demonstrated either in the cyst or in the gall-bladder in any of the cases described. Swain, however, is said to have felt one at his cannula tip at aspiration.²

Difficulty has been found in explaining the formation of these so-called idiopathic cysts. The gall-bladder has been found normal in size in three-quarters of the cases,² while jaundice has been absent in at least eleven besides the present case. Saint,³ after reviewing cases in regard to etiology,

rejects the theory of obstruction of the exit to the duodenum as a cause of their formation, on the ground that the biliary passages above may be, as in this case, normal in size.

The postulation of a valve action at the entrance to the cyst would overcome, I think, this objection. Quite often a crescentic fold of tissue has been observed obscuring the upper opening.² The existence of obstruction and usually² a valve mechanism at the exit is generally accepted. The gall-bladder will then act like a pump on the principle of the Bramah press so widely applied in hydraulic machinery. The tension of the cyst wall will be raised to such a point that at times the exit is straightened out and bile flows again into the duodenum. The effort required to effect this will constantly tend to increase, as in a hydronephrosis, and fibrous-tissue walls² will not withstand this. One can imagine that if once a minute round sacculus develops in the wall of a collapsible duct, possibly from weakness in the muscular coat or from deformity due to the presence of a pancreatic rest or from failure of efficient fusion of foetal elements, it will tend steadily to get bigger. It should be noted that the cyst found in a foetus (Heiliger) was only 2.3×3 cm.—the smallest recorded—and therefore presumably at the beginning of its growth.

There is support for this theory of a double-valve action in the fact that when the pressure in the cyst is relieved by drainage these cysts rapidly shrink in size.^{2, 3}

Stoney⁴ found on re-opening the abdomen seven weeks after external drainage of a cyst of four-pints capacity that "there was now no evidence of a cyst or tumour in the abdomen and it was not possible even to recognize its collapsed wall". This shrinkage of the cyst helps to explain why a primary anastomosis of the cyst to the bowel has been so very much more successful than a preliminary external drainage followed by a secondary attempt at anastomosis, the results of which have been poor. It follows, too, that a large anastomotic opening is necessary. The cases where dilatation of the bile-passages above and cholangitis have occurred would be explained by a defective functioning of the valve at the cyst entrance.

CONCLUSIONS.

The operation of choice is the simple one of a primary anastomosis of cyst to gut, except where the cyst is so small and so free from adhesions as to be easily extirpated; even if there is infection of cyst contents the relief of pressure will help to prevent spread of infection to the liver. Anastomosis of the gall-bladder to the intestine would appear to be of little curative value, at any rate where the upper biliary channels are not dilated. If a simple external drainage is required by the very critical condition of the patient, the secondary anastomosis of cyst to bowel should not be delayed more than a week or two if possible.

REFERENCES.

- ¹ WILSON, *Jour. Amer. Med. Assoc.*, 1930, Aug. 9.
- ² CLARK, *International Clinics*, 1932, i. (Bibliography.)
- ³ SAINT, *Brit. Med. Jour.*, 1932, i, 232.
- ⁴ STONEY, *Irish Jour. Med. Sci.*, 1929, Dec.

A CASE OF GAS-CYSTS OF THE INTESTINE.

By F. C. PYBUS.

SURGEON, ROYAL VICTORIA INFIRMARY, NEWCASTLE-UPON-TYNE.

M. R. J., a female, 48 years of age, was admitted to the Royal Victoria Infirmary with pyloric obstruction. For fifteen years she had suffered from stomach trouble. For the first nine years the symptoms had been intermittent, there being short periods of freedom. During an attack there was pain half to one hour after food, the pain being epigastric and referred between the shoulders. It was relieved by food or alkalis. For the last few years the pain had been almost constant, unrelieved by food, had often been present at night, and each week she had a copious vomit. She had lost five stones in weight during the past eight years.

On EXAMINATION.—The patient was emaciated, and on abdominal inspection gastric peristalsis was obvious, but no further abnormality was detected. There was nothing unusual in the gastric analysis. X-ray examination showed calcified glands in the mesentery, extending through the mediastinum and almost the whole length of the neck. Twenty-four hours after a barium meal there was still a considerable residue in the stomach. Dr. S. W. Davidson noted that there was 'gas' between the liver and the right diaphragm, and the colon itself was full of gas. In view of the operative findings a re-examination of the radiogram shows the outlines of some of the cysts, though this is somewhat obscured by gas in the colon.

OPERATION.—Laparotomy was performed on Aug. 25, 1931. On opening the peritoneum it appeared at first sight as if there was a massive colloid carcinoma of the upper abdomen. Further inspection showed the condition to be due to gas cysts. The Registrar's description, taken at the time, fully describes the condition.

The falciform ligament of the liver, its adjacent peritoneum, and segments of the small intestine were the seat of numerous gas-containing cysts. The cysts were in bunches or clusters, looking like soap bubbles or like the cysts of a hydatid mole. They were transparent and collapsed with a 'pop' on puncture. Some of the cysts communicated with each other, others were isolated and varied in size from a pin-head to a cherry, but they were mainly in clusters. The clusters were distributed as follows:—

1. In the falciform ligament and the peritoneum on either side for about 2 in.
2. In the peritoneal coat of the small intestine, either singly or in grape-like masses, affecting the lower part of the jejunum and distributed as follows:—

1 ft. of affected bowel.

1 ft. of normal bowel.

About 6 in. of affected bowel.

1 ft. of normal bowel.

1 ft. of affected bowel.

A pedunculated grape-like mass with single stalk (*Fig. 371*).

1 ft. of normal bowel.

2 ft. of affected bowel.

3 in. of normal bowel.

1 ft. of affected bowel, followed by the remaining healthy intestine.

A photograph was obtained of the general mass (*Fig. 370*) and of an isolated segment of jejunum (*Fig. 371*). A portion of the cysts was removed for the museum, and for culture and microscopic examination. The stomach itself was grossly dilated and thick-walled, the pylorus stenosed and with an active ulcer beyond adherent to the pancreas. Gastrojejunostomy was performed, from which the patient made an uneventful recovery. In February, 1932, she appeared in perfect health, and had increased four stones in weight.

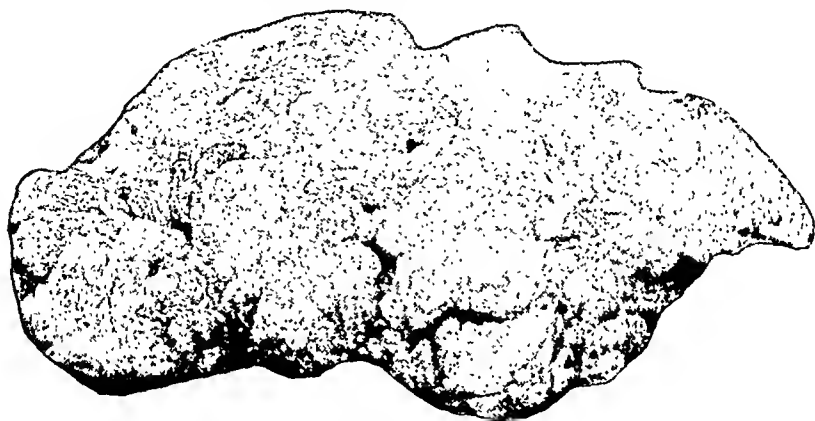


FIG. 370.—Coils of affected jejunum, showing gas cysts.



FIG. 371.—Segment of jejunum showing subserous cysts and a pedunculated portion.

PATHOLOGICAL REPORT (A. F. Bernard Shaw).—"The material consists of multiple round spaces lying in a loose connective tissue. The walls of the cavities appear to consist of the condensed surrounding tissue, and show no epithelial or serosal lining. In the connective tissue are many mobile histiocytes and some polymorphs."

On culture Dr. Slade reports that the cysts yielded a *Staphylococcus albus*, and a slender Gram-bacillus, with filamentous forms and characteristics of *Bacillus ramosus*.

RETROGRADE JEJUNOGASTRIC INTUSSUSCEPTION.

BY JUDSON T. CHESTERMAN.

THIS condition is of sufficient interest to warrant record, not only on account of its rarity, but also because of the ease of diagnosis if its possibility is remembered, and the hopeless prognosis without early surgical intervention.

HISTORY.—W. T., male, aged 31, was admitted to the Beckett Hospital, Barnsley, on March 15, 1933, complaining of severe abdominal pain. He was perfectly well till 8 a.m. that morning, when he was "suddenly doubled up" with severe epigastric colic while dressing. This was not relieved by alkaline powder, brandy, or turpentine stupes. At 9 a.m. he vomited a "teacupful" of bloody fluid and again at 10.30 about "a pint" of blood-stained fluid, but he obtained no relief. At 11.15 he was admitted to hospital and treated expectantly as "a medical colic". At 8.30 p.m. I was asked to see the patient, who had been in continuous unrelieved pain since admission. He stated that in 1926 he had been operated upon for "gastric ulcers", but the nature of the operation was unknown. He had had no symptoms of any abdominal disorder since.

ON EXAMINATION.—The patient lay doubled up on his left side; his face was ashen grey and he was perspiring freely. Pulse 64, temperature 98°, respirations 20.

On inspection the abdomen showed marked erythema due to the stupes; there was an old right supra-umbilical paramedian scar, and the whole outline of the stomach was clearly visible from the bottom of the bed. No rigidity was present on palpation, but the stomach outline was extremely tender. The stomach area was dull on percussion. Occasional sounds were noted in the right iliac fossa on auscultation. Rectal examination showed nothing abnormal. Atropine $\frac{3}{16}$ gr. was ordered to relieve the pain and facilitate examination.

At 8.50 p.m. there was a copious bloody vomit and at 9.15 p.m. the patient was re-examined. The stomach outline was not apparent, but was replaced by a round tumour in the left hypochondriac region, which was extremely tender, though the belly was soft elsewhere and palpation caused no discomfort.

The diagnosis of jejunogastric intussusception was made for the following reasons: (1) Sudden onset of severe epigastric colic; (2) Repeated bloody vomit; (3) Complete absence of rigidity and tenderness only over tumour.

OPERATION.—The abdomen was immediately opened by a left paramedian incision. Posterior no-loop iso-peristaltic gastrojejunostomy was found, with retrograde intussusception of 2 ft. of jejunum into the stomach through the stoma. Reduction after opening lesser sac. No cause found. The mesentery of the small gut was sutured to the transverse mesocolon near the jejunal border in two places to prevent recurrence.

SUBSEQUENT PROGRESS.—The patient was discharged on May 1 after an uneventful convalescence. On Aug. 4 he was at work and absolutely fit.

RECURRENCE OF GASTRIC CARCINOMA TWELVE YEARS AFTER PARTIAL GASTRECTOMY.

By A. B. PAIN,

LATE SURGICAL TUTOR, UNIVERSITY OF LEEDS, AND SURGICAL REGISTRAR,
GENERAL INFIRMARY AT LEEDS.

THIS case is of interest on account of the long interval which elapsed between the original operation and the appearance of the recurrence. An interval of twelve years, however, is by no means the longest on record: Shuman¹ recorded a case in which recurrence occurred seventeen years after partial gastrectomy, and Pearce Gould² recorded one in which recurrence took place eighteen years after partial gastrectomy, although he considered that in this case the history suggested that what was described as a recurrence was probably an entirely new growth. Peugniez³ reported a case in which there was clinical evidence of recurrence, probably in the stomach, twenty-four years after partial gastrectomy, although this suspicion was not confirmed by post-mortem or histological examination. Persson,⁴ in a series of 262 cases of carcinoma of the stomach submitted to partial gastrectomy, reported 9 cases which died of recurrence more than six years after operation. the longest interval being seventeen years.

Hartmann⁵ stated that recurrence of carcinoma more than two years after partial gastrectomy is exceptional, and, further, that almost all recurrences are lymphatic and peritoneal, recurrences in the gastric mucosa being very rare; he could only record 1 such case in 46 cases of proved recurrence. In a further report⁶ he recorded a series of 80 cases of gastric carcinoma which died with recurrences, all within four years of operation.

The history of the present case is as follows:—

The patient, a man of 40 years of age, was admitted to the General Infirmary at Leeds under the care of Mr. J. F. Dobson on July 5, 1919, with a six months' history typical of carcinoma of the stomach. There was a palpable tumour in the epigastrium to the right of the middle line; X-ray examination showed almost complete retention six hours after the ingestion of an opaque meal, with great dilatation of the stomach and almost complete absence of peristalsis; no filling defect was seen.

At operation, performed by Mr. Dobson on July 21, the stomach was found to be considerably dilated, with a large mass having the naked-eye characteristics of carcinoma occupying its pyloric half. Partial gastrectomy was performed, three-quarters of the stomach being removed, together with enlarged lymph-nodes along both curvatures; the jejunum was united to the stump of the stomach by an end-to-side retrocolic anastomosis. The man was discharged from hospital on Aug. 8, 1919.

No histological examination of the resected portion of the stomach was made, but Professor M. J. Stewart reported that the growth was a large soft carcinoma of the pyloric region.

The patient was re-admitted to the General Infirmary at Leeds on March 19, 1928, complaining that for four months previously he had suffered from epigastric pain coming on soon after meals, and that he had lost much

weight; he had also found that he had to take very small meals. Fractional test-meal examination showed that there was no free hydrochloric acid in the gastric juice, and that bile was present in all specimens. X-ray examination after an opaque meal showed no evidence of carcinoma in the remaining portion of the stomach, which emptied rapidly.

The patient was discharged on March 30, and from that time until May, 1930, was quite well. He then began to have pain and fullness after meals, and in September, 1930, he noticed a sense of obstruction to swallowing at the level of the lower end of the body of the sternum; he commenced to vomit after meals and rapidly lost weight.

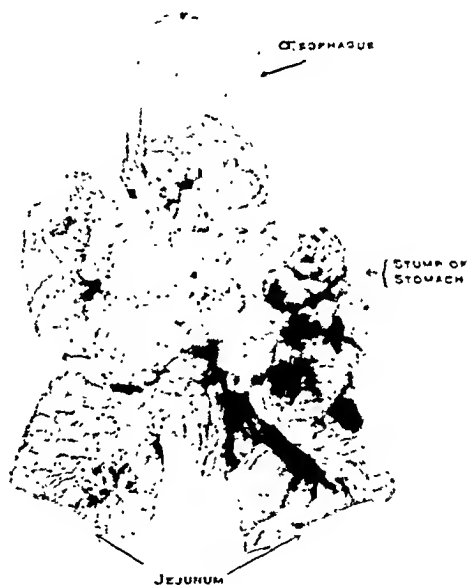


FIG. 372.—Condition found at autopsy.

He was re-admitted to the General Infirmary at Leeds on May 11, 1931, and again came under the care of Mr. Dobson. X-ray examination after an opaque meal demonstrated the presence of an obstruction at the cardiac orifice, with much dilatation of the œsophagus; the stump of the stomach could not be filled with bismuth for examination.

On May 26, under local anaesthesia, jejunostomy by Witzel's method was performed, using the efferent jejunal loop (A. B. P.). Death occurred four hours after operation.

POST-MORTEM EXAMINATION (Professor M. J. Stewart).—The small portion of the stomach (*Fig. 372*) which had been left behind was almost entirely converted into a large ulcerated carcinoma, situated saddle-wise across the upper part of the lesser curvature, completely encircling the gastro-jejunal

anastomosis, and extending at one point fully an inch up into the œsophagus. Only the region of the greater curvature in the middle of the remaining portion of the stomach had escaped. The tumour had a soft raised overhanging margin with various polypoidal projections, especially upwards into the œsophagus. Several glands heavily invaded by carcinoma lay in relation to the tumour; the growth was lightly adherent to the spleen at the upper end of the hilum, where it had penetrated the wall of the stomach. The pancreas was also slightly invaded, and there were two secondary nodules in the liver.

Histologically, the tumour was a fairly well differentiated adenocarcinoma, which had penetrated deeply into the muscularis mucosæ.

CONCLUSIONS.

In the absence of any histological examination of the first specimen, it is impossible to be dogmatic, but this seems to be a case of recurrence of gastric carcinoma in the stump of the stomach after partial gastrectomy. The fact that recurrence can take place after so long an interval suggests that the usual observation period of ten years is too short to be conclusive in assessing cures.

I am indebted to Mr. J. F. Dobson for permission to record this case, and to Professor M. J. Stewart for his help and for the use of the records of his department.

REFERENCES.

- ¹ SHUMAN, J. W., *Jour. Amer. Med. Assoc.*, 1926, lxxxvi, 1127.
- ² GOULD, E. PEARCE, *Brit. Jour. Surg.*, 1927, xv, 325.
- ³ PEUGNIEZ, P., *Bull. de l'Acad. de Méd.*, 1924, xci, 831.
- ⁴ PERSSON, M., *Ann. of Surg.*, lxxxvi, 321.
- ⁵ HARTMANN, H., *Bull. de l'Acad. de Méd.*, 1926, xcv, 44.
- ⁶ HARTMANN, H., *Ibid.*, 1932, cvii, 95.

APPENDICITIS IN A FEMORAL SAC, WITH ADVENTITIOUS VASCULARIZATION OF A SEGREGATED APPENDIX.

BY A. S. JOHNSTONE,

LATE SENIOR HOUSE SURGEON, LEICESTER ROYAL INFIRMARY.

DISJUNCTION of an appendix from localized destruction of the whole thickness of the organ is by no means uncommon, the vitality of the distal portion being ensured by the integrity of that portion of the meso-appendix. In the following case the solution of continuity was shared alike by the appendix and its mesentery.

HISTORY.—A moderately well nourished, healthy multipara, aged 67, was admitted to hospital complaining of a swelling in the right groin of seven weeks' duration. Except for enteric fever some forty years previously, she had had no important illness, no previous attack of appendicitis, and was not aware of the existence of a femoral hernia.

Three months previously she had severe colicky pains about the umbilicus. She had no vomiting and passed a restless night. On the following day, though the general malaise continued and was associated with anorexia, she had a normal evacuation of the bowels and was able to carry out her daily household work. A week later discomfort in the right groin called her attention to a swelling which appeared inflammatory, and after repeated fomentation an abscess opened through the skin and discharged a large quantity of foul-smelling pus. Thereafter, the swelling having subsided, the abscess quickly healed. A month later a painless swelling reappeared at the site of the abscess, and as it gave some inconvenience in walking operation was decided on.

ON EXAMINATION.—The contour of the abdomen was normal, there was neither hyperæsthesia nor tenderness, and no muscular rigidity. In the right femoral triangle lateral to the pubic spine was a lobulated cystic swelling some 35 mm. in diameter, apparently fixed to the deep fascia at the femoral ring, though free from the skin, which over it was of a slightly bluish tint. There was no impulse on coughing, and the cysts could not be emptied by digital pressure. There were no enlargements of adjacent lymph-nodes, no varicose veins, and no cutaneous source of lympho-vascular infection.

OPERATION.—Under general anaesthesia, after longitudinal incision, the skin was reflected bilaterally. Some cysts opened during the delimiting dissection allowed the escape of a mucinous material, and beyond the cystic area was exposed the wall of a sac which could be traced to the femoral ring. Very dense adhesions were present, and considerable difficulty was experienced in separating the sac from the femoral vessels.

After isolation the sac was opened and the appendix was found attached to its anterior surface, the base of the appendix towards the neck of the sac, to which parts the mucoid cysts were closely related. A strand of great omentum attached to the appendix was divided, but the caecum could not be felt by the finger passed through the femoral canal. The sac and appendix were then removed and the canal closed.

MACROSCOPIC FINDINGS.—Inspection of the portions removed showed a multilocular cystic mass close to the base of the appendix with the lumen of which it communicated through a small perforation. Lying within the lumen of the appendix at this point were two small chips of china, probably of etiological importance as regards the mucinous cysts. It was evident that the appendix had an adequate blood-supply through its adventitious adhesion to the wall of the sac.

COMMENTARY.

The interest of this case lies in the occurrence of an auto-appendicectomy and the continued viability of the appendix after the acquisition of a new blood-supply. It may be surmised that there was a pre-existing femoral sac in which the appendix had come to lie at some earlier period, and, after adhering to the wall, had acquired a new blood-supply. It is unlikely that this could have occurred during suppuration, when considerable thrombosis and phlebitis in the meso-appendix would be present. The lumen of the

appendix was found obliterated and entirely fibrous at its proximal end, where lay the two foreign bodies. An acute attack of obstructive appendicitis occurred, passed to abscess formation with gangrene at the constricted area, and finally caused complete severance of the appendix. It seems unlikely that the appendix was detached prior to the attack, as the foul-smelling pus indicated that faecal infection had been present. A small strand of omentum was all that remained as a barrier between the infected area and the abdominal cavity. After resolution of the abscess the appendix returned to a fairly normal condition in its new surroundings, its blood-supply being adequate. A small perforation existed in the wall adjacent to the foreign bodies whose presence excited a continued secretion of mucus, which, finding its way into the surrounding tissues of the sac wall, led to the formation of multilocular cysts.

Perusal of the literature of femoral appendicitis fails to discover a similar occurrence. A number of cases of acute appendicitis in a femoral hernial sac are described and cases of resolution are recorded, but the occurrence of auto-appendicectomy and the survival of the separated part of the appendix appears unique.

I am indebted to Mr. Bolton Carter, of Leicester, for permission to publish this case, and to Mr. D. M. Greig for his assistance in preparing it.

REVIEWS AND NOTICES OF BOOKS.

Rose and Carless' Manual of Surgery. By CECIL P. G. WAKELEY, D.Sc. (Lond.), F.R.C.S. (Eng.), F.R.S. (Edin.), Surgeon, King's College Hospital, etc.; and JOHN B. HUNTER, M.C., M.Chir. (Cantab.), F.R.C.S. (Eng.), Surgeon, King's College Hospital, etc. Fourteenth edition. Large 8vo. Pp. 1487 + viii, with 721 illustrations and 21 plates (16 coloured). 1933. London: Baillière, Tindall & Cox. 30s. net.

It is only three years since the appearance of the last edition of this work, which has now become the students' and practitioners' classic. It is a testimony both to the diligence of the editors and to the progress of surgery that so much addition and alteration have had to be made in the present issue. Hitherto an attempt has been made to keep the work to the size of a 'handbook', but now, although this single-book edition is presented and the total number of pages not increased, yet the alternative of a double volume is offered at the same price.

As regards general get-up, there has been a marked advance. The paper is of a superior quality and surface, so that the reproduction of the figures leaves nothing to be desired. Of the illustrations no fewer than 300 out of a total of 721 are new, and about half the plates are either new or redrawn. In the selection of additional figures, the editor has very wisely been guided by the main consideration of clearness and instructive character. Thus line drawings and diagrams have been put in rather than photographs. These three points—the new paper, the new figures, and the large number of line drawings—in themselves constitute a notable advance. The X-ray plates are now interspersed where wanted in the text instead of being placed all together at the end of the book.

Among the sections which have been largely revised or re-written are the following: the treatment of malignant disease by radium; the surgery of the sympathetic nervous system; thoracic surgery; and the treatment of fractures according to Böhler's technique.

A work of this size and importance requires the co-operation of several specialists. Not only has Mr. Wakeley been joined by Mr. Hunter as co-editor, but he has had the assistance of Carnegie Dickson (pathology), V. E. Negus (ear, throat, and nose), Bishop Harman (ophthalmology), Eardley Holland (gynaecology), Sir Frank Connor (military surgery), and C. F. Hadfield (anaesthesia).

It is a very happy thing that the original work of two great King's College surgeons should be carried on by two of the King's men of to-day, and that in building up this great work on surgery they should be perpetuating the memory and work of the greatest of British surgeons, Lord Lister of King's. We congratulate the editors and publishers on a notable success.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, etc. Fourth edition, revised and enlarged. Large 8vo. Pp. 287 + xx, with 335 illustrations, some of which are in colour. 1933. Bristol: John Wright & Sons Ltd. 21s. net.

This is a new and improved edition of what has now become a very well-known text-book. A strong point of this work has always been the number and excellence of the illustrations, and in this particular the present edition excels.

Some of the illustrations are works of art, and, as was once said about Howard Kelly's extravagantly illustrated work on appendicitis, "any one of them might be

framed and hung up to decorate the office". Pictures in colour are sometimes misleading, but the figure illustrating rupture of the urethra is very realistic indeed. Many of the simpler diagrams are very telling, especially those that have been made in elucidation of actual cases; they supply excellent models for anybody interested in the keeping of simple yet graphic case records.

Of course, there are a few things which strike the reviewer and suggest mild criticism, but they are not really very important. For instance, for testing surface temperature, the illustration suggests that the tips of the fingers are sufficient, whereas one has always recognized that it is much wiser to use the palm of the hand. But the book is indispensable and there can be nothing but praise for the continued efforts of the author and his publishers, not only to keep it up to date, but to improve it.

The Practice of Surgery. By RUSSELL HOWARD, C.B.E., M.S. (Lond.), F.R.C.S., Surgeon, London Hospital, etc.; and ALAN PERRY, M.S. (Lond.), F.R.C.S., Surgeon, London Hospital, etc. Fourth edition. Medium 8vo. Pp. 1338 + viii, with 584 illustrations and 8 coloured plates. 1933. London: Edward Arnold & Co. 30s. net.

THE new edition of this well-known book maintains the characters which made former editions so popular. Its merits are many. In the first place it is a student's text-book and has remained so. The authors have selected with much discrimination what is really necessary to teach the undergraduate. In the second place, having made up their minds on this supremely important question, they have stated simply, directly, and sometimes dogmatically what should or should not be done under any given circumstances. This is what the student requires when such debatable subjects as the operative treatment of exophthalmic goitre or chronic mastitis are in question. Taken as a whole the recommendations are those usually accepted by surgeons of experience.

There are a few—very few—omissions, such as the treatment of Raynaud's disease by sympathetic ganglionectomy, and a few incorrect statements, such as that which leads the reader to believe that temporary recovery only has followed Trendelenburg's embolectomy. Also we read the trans-sacral removal of the rectum has been almost entirely abandoned, whereas there has of late been increased interest in this operation both in England and abroad.

Gastrojejunostomy for hæmorrhage from a gastric ulcer recommended on page 715 is worse than useless, as it exhausts the patient and does not stop the bleeding. There are better things to do not mentioned in this text-book. It is also not quite correct to say that Continental surgeons recommend partial gastrectomy for duodenal ulcers. Some surgeons, both in this country and abroad, recommend partial gastroduodenectomy.

We doubt if Phelps' operation for talipes is often carried out nowadays, and the suggestion that Marsh's knee truss for internal derangement keeps the semilunar cartilage in place is not in conformity with pathology. However, there is very little to cavil at in this text-book. There is rather a clumsy sentence on page 299 and another on page 1265. The printing and illustrations are excellent. The work bears the imprint of a great teacher: its simple, direct, and well-balanced statements render it pre-eminently suitable for the undergraduate student, by large numbers of whom it will doubtless be read.

Operative Surgery. By WARREN STONE BICKHAM, M.D. and Phar. M. (Tulane), M.D. (Columbia), F.A.C.S., Former Surgeon in charge of General Surgery, Manhattan State Hospital, New York, etc.; and CALVIN MASON SMYTH, jun., B.S., M.D., F.A.C.S., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania, etc. Vol. VII, The Newest Operations and General Index. Royal 8vo. Pp. 819 + viii, with 765 illustrations. 1933. Philadelphia and London: W. B. Saunders Co. 55s. net.

THIS is a seventh and supplementary volume to the original work in six volumes published in 1924. Since that time many additions have been made to the operations which surgeons may be called upon to perform, and this volume has become

necessary. It is divided into sections, each of which modifies and brings up to date a section in the complete work. There is an extremely well-thought-out introduction concerning the art and craft of surgery at the present time. It is followed by sections dealing with pre- and post-operative treatment, with sterilization, with anaesthesia, in which basal and local anaesthesia are most fully considered, and with electrosurgery.

In the sections dealing with the chest and colon the most numerous additions are to be found. The chest section in particular is especially full, which is to be expected when we remember that until recently this was practically a virgin field. More might have been made of the surgery of the sympathetic nervous system. The description of cervical sympathectomy is vague and incomplete, and removal of the inferior mesenteric ganglion in the abdomen is not mentioned at all. It may be that this branch of surgery is not so popular in America as it is in this country.

In places the originators of new operations are not given, but the author apologizes for this. An occasional lapse in this direction is hardly to be avoided when almost all the procedures are taken from descriptions in current medical literature.

The illustrations are numerous and clear, and they show most of the operations step by step. The text is clear and easily readable. The addition of this volume certainly makes it the most complete and up-to-date work on operative surgery in the English language.

The Art of Surgery: A Text-book for Students and Practitioners. By H. S. SOUTAR, D.M., M.Ch. (Oxon.), F.R.C.S., Hon. M.D. Trinity College, Dublin, Surgeon, London Hospital. Second edition. Large 8vo. Pp. 630 - x. Illustrated. 1933. London: William Heinemann (Medical Books) Ltd. 30s. net.

THE first edition of this work appeared in 1928. We are assured in the seven-line preface to the second edition that "the greatest care has been taken to include all that is valuable in recent methods", but we confess that we have been rather disappointed in the fulfilment of this promise. For example, neither the sympathetic nervous system nor Raynaud's disease is mentioned in the index, nor is any reference to sympathectomy made in the article on Hirschsprung's disease.

The book certainly has a charm of its own, in its differences from other text-books. Its wide margins, in which appear the many little 'thumb-nail' sketches, present an invitation to the student to add notes of his own with further drawings. Many of the coloured figures, although undoubtedly works of art, do not convey a clear idea of the thing presented.

In pointing out some of the defects of this book, we do not wish to do anything but improve it. There is a great charm in its get-up, in its diction, and its well-balanced choice of what to put in and what to leave out.

Recent Progress in Medicine and Surgery, 1919-1933. Edited by SIR JOHN COLLIE, C.M.G., M.D., D.L., J.P. With a Foreword by LORD HORDER OF ASHFORD, K.C.V.O., M.D., F.R.C.P. Demy 8vo. Pp. 368 + xii, with 34 illustrations. 1933. London: H. K. Lewis & Co. Ltd. 16s. net.

It is useful to take stock of one's knowledge from time to time, and this is especially the case with medicine and surgery, where progress just now is extraordinarily rapid. New discoveries and new inventions have led to a new nomenclature and the use of terms which the older members of the profession can only understand by their etymology—and not always then. The modern school often uses words which seem to be composed of corrupt Greek and mediæval Latin. Sir John Collie has enlisted the services of an excellent team of writers to deal with recent progress in medicine and surgery, and has himself contributed an article on active immunization against diphtheria, a subject in which he has interested himself as Chairman

of the Public Health Committee of the Paddington Borough Council. Mr. H. A. T. Fairbank, D.S.O., Senior Orthopædic Surgeon to King's College Hospital, writes on orthopædic surgery and its modern development as a result of experience in the European War. Mr. J. Swift Joly, who is Senior Surgeon to St. Peter's Hospital, describes, in his article on urology, the great advances which have been rendered possible by the use of X rays and the revolution in the treatment of the prostate which has taken place within the last twenty years. The conservative treatment of surgical tuberculosis has been entrusted to the capable pen of Sir Henry Gauvain, and that on plastic surgery to Sir Harold Gillies, who has made the subject his own. The articles on manipulative surgery are written by Dr. J. B. Mennell, who proves that there are better 'bonesetters' within the profession than amongst those who work without a registrable qualification; whilst Mr. I. W. Magill tells of the very great changes which have occurred in the production of anæsthesia since the time when chloroform was administered on a towel or a square of lint. The advances in medicine have been placed in equally capable hands, and amongst the special departments there are articles on the eye, oto-rhino-laryngology, and bacteriology, written respectively by Sir William S. Duke-Elder, Sir James Dundas-Grant, K.B.E., and Dr. R. Tanner Hewlett. Professor J. M. Woodburn Morison has a well-illustrated article on radiology which is thoroughly practical.

Surgical Anatomy. By C. LATIMER CALLANDER, A.B., M.D., F.A.C.S., Assistant Clinical Professor of Surgery and Topographic Anatomy, University of California Medical School. With a Foreword by DEAN LEWIS, M.D., Sc.D., LL.D., F.A.C.S. Super royal 8vo. Pp. 1115, with 1280 illustrations, some in colour. London and Philadelphia: W. B. Saunders Co. 63s. net.

THIS is not merely a treatise on surgical anatomy; in fact, in places descriptive anatomy is dealt with less thoroughly than its application. Many operations—and these not always the most important in surgery—are dealt with at length; other operations are briefly touched upon with no particular attempt to emphasize their relation to the anatomy of the region then under discussion. But, as suggested, the book contains a great deal more than its title would indicate. There are in places full accounts of the signs and symptoms of different surgical diseases and injuries, and, last but by no means least, it is profusely and beautifully illustrated. The figures themselves more than justify the book. They are on the whole well chosen, and as the keynote throughout is operative surgery they demonstrate the relation of anatomy to the operation under discussion.

The book is written in a diffuse, digressive manner, rather typical of American productions, and for this reason it is not likely that the letterpress will make a strong appeal to British surgeons; but there is no volume of surgical anatomy in this country which has anything like the illustrations which are to be found over the name of Ralph Sweet and others. As is usual in the case of work coming from the Saunders Company, the general output of the volume is first rate, though perhaps one might take exception to one minor point—the extreme weight of the volume.

A Short History of Surgery. By SIR D'ARCY POWER, K.B.E., F.R.C.S., Hon. Librarian of the Royal College of Surgeons of England. Crown 8vo. 1933. London: John Bale, Sons & Danielsson, Ltd. 3s. 6d. net.

THIS is a delightful little book which gives in its eighty odd pages a good account of the subject with which it deals. It will doubtless become the regular primer of the history of the healing art, for it is written in the easy style which we associate with Sir D'Arcy Power and it is all made so interesting that there will be few who put the book down before completing its pages.

The headings of the chapters give a good indication of the way the author deals with his task. Those on Specialism, American Surgery, and Surgical Nursing indicate the wide scope of this work. The author has a good word to say for what

we now look upon as the very out-of-date type of nurse, and instances the famous Mrs. Porter—but she was surely the light of her calling in the Royal Infirmary in Edinburgh, and not in charge of Lister's operation ward in Glasgow as stated. These wonderful women may have been ill-educated and a little unclean, but as one who is able to judge (G. H. Edington) has written, "they knew how to nurse and they did it".

Another little error has crept into the chapter on Modern Surgery, in which the author deals so succinctly with the development of the treatment of wounds, for he states that the crude carbolic which was brought to the notice of Lister by Thomas Anderson, Professor of Chemistry in Glasgow, had been used to prevent decomposition in the refuse heaps of that city. As a loyal Englishman one has always thought that it was its remarkably beneficial effect on the sewage of Carlisle that really gave the inspiration to the use of carbolic acid!

The chapter on Specialism is wonderfully complete, including as it does a paragraph on dentistry. Despite the shortness of the chapter devoted to American Surgery, it is rather sad to find no mention of McDowell and his pioneer ovariectomy.

Every student should have the opportunity of reading this little book, and it would not be a bad thing to make its perusal introductory to the study of surgery.

The book is of handy form and well printed on good paper, and is provided with a useful index.

Papers Relating to the Pituitary Body, Hypothalamus, and Parasympathetic Nervous System. By HARVEY CUSHING, Professor of Surgery (Emeritus), Harvard University, etc. Large 8vo. Pp. 234 — viii, with 99 illustrations. 1933. London: Baillière, Tindall & Cox. 29s. net.

FOUR lectures delivered by Professor Harvey Cushing during the past three years, all dealing with the physiology and pathology of the interpeduncular region of the brain, and previously scattered in different medical journals, are now happily collected together in this volume. Two are concerned primarily with the pituitary body, and two with the hypothalamus as a cerebral centre of the parasympathetic system. The pituitary lectures are clearly the fruit of many years of study and experience; the others, though equally absorbing and stimulating, are more conjectural.

In the Lister Memorial Lecture on "Neurohypophysial Mechanisms from a Clinical Standpoint", Professor Cushing presents a masterly review of the experimental and clinical evidence of the interdependence of the anterior lobe of the pituitary body, the infundibular lobe with its epithelial investment, and the diencephalon. The lecture reads almost like the report of the proceedings in a court of law, the author adopting successively the rôles of witness, counsel, and judge. It is left to the reader as a member of the jury to decide whether the mechanisms which govern metabolism, the water balance of the body, the functions of sex, and even the perpetuation of the species, reside primarily in the pituitary body or in the interbrain.

The lecture on "The Basophil Adenomas of the Pituitary Body" describes the steps which led to the discovery that patients showing a peculiar form of adiposity, associated with kyphosis, sexual dystrophy, hypertrichosis, high blood-pressure, erythremia, and weakness—a clinical picture which has already been named 'Cushing's syndrome'—have basophilic adenomas. The clinical and pathological findings are correlated in 16 cases, some observed personally and the remainder reported by others. The picture resembles that produced by some adrenal tumours, and even when a basophilic adenoma has been found in the pituitary body, changes in the other ductless glands have been present also. It is argued, however, that the pituitary lesion is the primary one, the changes in the other endocrine organs being secondary.

The very remarkable experiments recorded in the lecture on "The Posterior Pituitary Hormone and the Parasympathetic Apparatus" were originally intended merely to establish the existence of a posterior-lobe hormone. As a result of the

experiments, however, the fact emerges that the effect of injecting pituitrin intramuscularly or intravenously is entirely different from the effect produced by intraventricular injection. Whereas the former resembles the reactions of sympathetic stimulation, intraventricular injection produces a reaction which is predominantly parasympathetic, resembling closely the response to intraventricular pilocarpine, being counteracted by atropine, and not occurring when there is internal hydrocephalus or disease of the hypothalamus. It is therefore suggested that pituitrin stimulates parasympathetic centres in the hypothalamus. The lecture deserves careful study and will provide much food for thought. Some of the reactions described appear to be a peculiar mixture of sympathetic and parasympathetic responses, and it may be that stimulation of the hypothalamus, if this is indeed what occurs, gives rise to phenomena which are neither purely parasympathetic nor sympathetic in nature.

This difficulty in the interpretation of the evidence recurs in the last lecture on "Peptic Ulcer and the Interbrain". Records are quoted of cases in which ulceration of the stomach and duodenum has occurred in association with lesions of the brain, and it is suggested that the ulcers may be caused by hyperacidity and localized vascular disturbances in the stomach resulting from stimulation of parasympathetic centres in the hypothalamus. In the majority of the cases the cerebral lesion was neoplastic, but Professor Cushing goes so far as to hint that the centres in the interbrain might be affected by psychic influences, the result being a chronic gastric or duodenal ulcer. It is a fascinating theory, and it is most stimulating to meet an Emeritus Professor with the imagination of perpetual youth! We are left in doubt why psychic stimuli should evoke from the hypothalamus sometimes sympathetic and sometimes parasympathetic responses, but we hope that further investigations along the lines indicated in the lecture may provide the solution of this intricate problem.

The lectures are profusely and clearly illustrated, and enough has been said to indicate that they may be read and re-read with much profit.

History of Urology. Prepared under the auspices of the American Urological Association. Editorial Committee: E. G. BALLENGER, W. A. FRONTZ, H. G. HAMER, B. LEWIS. Two volumes. Royal 8vo. Vol. I. Pp. 386 + xii, with 46 illustrations. Vol. II. Pp. 362 + vii, with 12 illustrations. 1933. London: Baillière, Tindall & Cox. 36s. net.

THIS is a useful and comprehensive history of one of the later specialties of surgery. It is more valuable because very full references are made to each of the subjects with which it deals. The book has been prepared under the auspices of the American Urological Association by a small editorial committee with Dr. Bransford Lewis as Secretary. Each volume contains fifteen chapters by various writers of repute in their own subjects, the articles ranging from surgical diseases of the kidney to affections of the scrotum, testes, and seminal vesicles. There are also chapters on the anatomy and physiology of the genito-urinary organs, anaesthesia, diagnosis, bacteriology, and the neuroses and functional disorders of the genito-urinary tract. All are of great practical value to surgeons practising genito-urinary surgery, and where all are so good it is perhaps invidious to draw attention to the particular excellence of the chapters on prostatic surgery. The first eight chapters of the first volume deal with the early history of urology in the different cities and states of the Union. They are good reading because they are full of details showing the varying social conditions under which medical practitioners lived even as lately as 1900: men of the highest culture in cities like New York, Philadelphia, Boston, and Baltimore; others practising in outlying parts who had received no medical education and were under conditions not far removed from those pictured by Bret Harte, who owed their very existence to their ability to shoot at sight. Yet both classes did good work in urology, and from their pioneer labours came the American Urological Association, "born of poor but honest parentage in a humble wine house of New York on Washington's Birthday, February 22nd, 1902". Even if the book had no other value, it would be well worth reading for its pictures of a time which is now beyond recall, though it is so near that the authors write from personal knowledge of the great originals whose stories they tell.

The Physician as a Man of Letters, Science, and Action. By THOMAS KIRKPATRICK MONRO, M.A., M.D., Regius Professor of Medicine in the University of Glasgow. Demy 8vo. Pp. 212 + viii. 1933. Glasgow: Jackson, Wylie & Co. 10s. 6d net.

DR. MONRO, the Regius Professor in the University of Glasgow, gives a charming and useful account of members of the medical profession who have attained distinction in other fields of work than those for which they were intended. A few were saints, several were mystics, many were poets, others excelled as writers. Not a few were rascals, and of these a due proportion were caught and hanged. It is noteworthy that until recently the few peerages were bestowed on those who, deserting medicine, had excelled as lawyers. Professor Monro gives a short but sufficient account of each class, and his book will long survive as a work of reference by those who wish to show how a well-trained doctor can turn his hand successfully to many different branches of work. Another edition will certainly be asked for in a short time. A few of the omissions may then be made good. There might be an account of John Smith, who wrote the *Sacred Anatomy, or King Solomon's Portraiture of Old Age*, showing that all anatomy was taught in the first six verses of the twelfth chapter of Ecclesiastes (this is still good reading); of Dr. Marsden, who founded the Royal Free Hospital; of William Kitchen Parker, Wesleyan preacher, general practitioner, and morphologist; of 'Stone henge', who turned from medicine to dogs; and of Augustine Henry, who abandoned medicine, made his name as a botanist in China, and ended as Reader in Forestry at Cambridge and in Dublin.

In some instances Professor Monro is able to attribute honour where honour is due to those whose work was in danger of being forgotten. He shows that Charles Morrison was transmitting messages by electrified wire as early as 1753, and that Henry Faulds published a method of identification by finger-prints in 1880. The book concludes with an account of some eminent American and European doctors who have made their names in other fields of knowledge, and of a few Englishmen who began as medical students but never qualified.

The Technic of Local Anæsthesia. By ARTHUR E. HERTZLER, A.M., M.D., Ph.D., LL.D., F.A.C.S., Professor of Surgery in the University of Kansas, etc. Fifth edition. Large 8vo. Pp. 292, with 148 illustrations. 1933. London: Henry Kimpton. 25s. net.

THIS very delightful book has now reached its fifth edition, and is dedicated to Edwin Edward Wuttke, M.D., for five years Resident of the Halstead Hospital, "a veteran of two wars—in war or in peace a true soldier always". It begins with an account of drugs used in local anæsthesia, but omits some of the more recent and popular narcotics in use in this country. Chapter II describes the technique of injection; the author does not describe the short Schimmel needle, or the powerful syringe such as we in England prefer; he emphasizes the essential need for sharp and fine needles. In subsequent chapters he describes individual operations in great detail, particularly those concerned with the fifth nerve. His descriptions of paravertebral and splanchnic injection and of sacral blocking and trans-sacral injection are excellent.

Spinal anæsthesia is described in Chapter XIV by Arch. E. Spelman, and is very well done, the photographs illustrating it being clear and most useful. Raymond F. Gard writes of intravenous anæsthesia. He omits any mention of our old friend hedonal, and confines himself to sodium amylal (one of the many barbiturates).

As would be expected, the illustrations are numerous—no fewer than 148 in a book of 290 pages. They are all excellently drawn and one regrets no acknowledgment is made to the artists.

The author in his preface to this edition stresses the fact that the "work differs essentially from other books on local anesthesia in that it recommends the use of minimum amounts of solutions, not because of safety, but because a more accurate technic is possible than when large amounts of liquids are injected haphazardly. It advocates infiltrative anesthesia rather than regional blocking because constriction of vessels is secured thereby which aids in exact anatomic operating."

There is no book on local anæsthesia more clearly written and more beautifully illustrated, and none so charmingly "printed and papered".

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

- Text-book of Pathology.** By ROBERT MUIR, M.A., M.D., Sc.D., LL.D., F.R.S., Professor of Pathology, University of Glasgow, etc. Third edition. Demy 8vo. Pp. 957 + viii, with 546 illustrations. 1933. London: Edward Arnold & Co. 35s. net.
- Die Embolie.** By Dr. SIGURD FREY (Königsberg). Royal 8vo. Pp. 178, with 36 illustrations. 1933. Leipzig: Georg Thieme. RM. 12.
- Oral Surgery.** By STERLING V. MEAD, D.D.S., M.S., Professor of Oral Surgery and Diseases of the Mouth, and Director of Research, Georgetown University Dental School, etc. Royal 8vo. Pp. 1087, with 403 illustrations. 1933. London: Henry Kimpton. 63s. net.
- Operative Surgery: the Abdomen and Rectum.** By Dr. MARTIN KIRSCHNER (Tübingen). Authorized translation by I. S. RAYDIN, B.S., M.D., J. William White Professor of Research Surgery, University of Pennsylvania. Super royal 8vo. Pp. 569 + xiv, with 395 illustrations, mostly coloured. 1933. Philadelphia and London: J. B. Lippincott Company. 50s. net.
- Fractures.** By PAUL B. MAGNUSON, M.D., Associate Professor of Surgery, Northwestern University Medical School, Chicago. Medium 8vo. Pp. 466 + xx, with 317 illustrations. 1933. Philadelphia and London: J. B. Lippincott Company. 25s. net.
- Surgical Anatomy.** By GRANT MASSIE, M.B., M.S. (Lond.), F.R.C.S. (Eng.), Assistant Surgeon, Guy's Hospital, etc. Second edition. Medium 8vo. Pp. 458 + x, with 147 illustrations, many in colour. 1933. London: J. & A. Churchill. 18s. net.
- Die Luft- und FetteMBOLIE.** By Dr. Med. SIEGFRIED HOFFHEINZ (Leipzig). Vol. 55 of *Neue Deutsche Chirurgie*. Royal 8vo. Pp. 259 + xii, with 50 illustrations. 1933. Stuttgart: Ferdinand Enke. Paper covers, RM. 20; bound, RM. 31.20.
- Manuel de Radiodiagnostic clinique.** By R. LEDOUX-LEBARD (Paris). Royal 8vo. In two volumes. Pp. 1075 + xvi, with 1143 illustrations. 1933. Paris: Masson et Cie. Fr. 260.
- Frakturen und Luxationen: ein Leitfadens für den Studenten und den praktischen Arzt.** By Prof. Dr. GEORG MAGNUS, Leitender Arzt der chirurgischen Abteilung des Krankenhauses bergmannsheil in Bochum. Second edition. Large 8vo. Pp. 86, with 43 illustrations. 1933. Berlin: Julius Springer. RM. 3.60.
- Human Embryology and Morphology.** By Sir ARTHUR KEITH, M.D., F.R.S., LL.D., D.Sc., F.R.C.S., Master of the Buckton Browne Research Farm. Fifth edition. Medium 8vo. Pp. 558 + viii, with 535 illustrations. 1933. London: Edward Arnold & Co. 32s. 6d. net.
- An Introduction to the Study of the Nervous System.** By E. E. HEWER, D.Sc. (Lond.), Reader in Histology in the University of London, etc., and G. M. SANDER, M.B., B.S. (Lond.), F.R.C.S., Surgeon, London Lock Hospital for Women and Children, etc. Second edition. Large 8vo. Pp. 147 + xiv, with 65 illustrations. 1933. London: William Heinemann (Medical Books) Ltd. 21s. net.
- Allgemeine und spezielle chirurgische Operationslehre.** By Dr. MARTIN KIRSCHNER (Tübingen). Vol. V/1. Die operative Beseitigung der Bauchbrüche (M. KIRSCHNER). Die Eingriffe an den weiblichen Geschlechtsorganen (G. A. WAGNER). Royal 8vo. Pp. 395 + x, with 304 illustrations. 1933. Berlin: Julius Springer. Paper covers, RM. 78; bound, RM. 86.
- Urinary Infections.** By CLIFFORD MONSON, O.B.E., F.R.C.S., Surgeon to St. Peter's Hospital for Urinary Diseases, etc. Fcap 8vo. Pp. 76. 1933. London: John Bale, Sons & Danielsson, Ltd. 2s. 6d. net.
- A Practical Medical Dictionary.** By THOMAS LATHROP STEDMAN, A.M., M.D. Twelfth edition. Royal 8vo. Pp. 1256 + xii. Illustrated. 1933. London: Baillière, Tindall & Cox. 35s. net.

THE BRITISH JOURNAL OF SURGERY

VOL. XXI.

APRIL, 1934.

No. 84.

IPSISSIMA VERBA.

By SIR D'ARCY POWER, K.B.E., LONDON.

I. LISTER AND WIRING THE PATELLA.

THE memory of Lord Lister as the introducer of aseptic surgery is immortal. The memory of Lister as a bold and successful surgeon is already perishing. In 1870 the treatment of transverse fracture of the patella was beginning to attract attention. The older practice was to put the patient to bed, raise the injured leg on an inclined plane to relax the quadriceps muscle, and allow fibrous union to take place—a matter of six to eight weeks. The result was a gap of varying size which tended to increase with use. The leg, however, was useful and the patient was able to follow even so trying an occupation as that of an omnibus conductor at a time when he had to stand for long hours on a 'monkey board' holding on by a strap. More daring surgeons sought to get closer union by means of Malgaigne's hooks—a pair of sharp-pointed double hooks connected by a spring. They were inserted through the skin into the upper and lower fragments. The punctures often suppurated, the joint became involved, and the leg had to be amputated in the lower third of the thigh. A few adventurous surgeons brought the fragments together by a subcutaneous circumferential suture of silver wire.

It was a great experiment, therefore, and worthy, his contemporaries thought, of the utmost reprobation when Lister opened the knee-joint and passed a stout silver wire through the two fragments. He says that he had long been looking for a fractured patella with the intention of wiring it, but had been anticipated by [Sir] Hector Cameron, a former house surgeon and then Surgeon to the Glasgow Infirmary, who performed the operation successfully and without suppuration on March 5, 1877.

"In October of the same year," Lister continues, "a patient with transverse fracture of the patella was admitted under my care in King's College Hospital. He was a man of forty years of age, who, while riding on horseback, had his horse stumble and fall. He was thrown over the horse's head, falling on the right knee. He could not rise and was brought to the hospital. In the first instance, I attempted with this patient to bring the upper fragment down, so that it should be in contact with the lower. For

this purpose I applied an apparatus into the details of which I need not enter further than to say that it was so arranged that the upper fragment by means of weights and pulleys was drawn down. Four days later, however, I found that there was still a quarter of an inch interval between the fragments, and I suggested to the patient the operation of cutting down and applying the wire suture. This, however, he would not then consent to and preferred returning home to be under the care of his ordinary medical attendant. Eight days later—or fourteen days after the accident—he was readmitted, expressing a wish to be operated upon.

“On 26th October I accordingly proceeded to operate, making a vertical incision about two inches in length over the patella exposing the fragments which were then one inch apart. My inability to bring down the upper fragment into contact with the lower became explained when the parts were exposed; for there were found between the fragments extremely firm coagula with fibrous tissue, fascial and periosteal, mingled with them, constituting so firm a mass as to make it quite impossible for the two fragments to be brought into contact. The clots having been completely cleared away from between the fragments and from the interior of the joint, I applied a common bradawl in the middle line of the patella drilling each fragment obliquely so as to bring out the drill upon the broken surface a little distance from the cartilage. Pretty stout silver wire was then passed through the drilled openings and the fragments thus strung upon it were pushed firmly home and so brought accurately into apposition.

“Before they were brought together, however, an arrangement was made for the drainage of the joint. This was done on the same principle in all the cases that I have to record and I may therefore describe the matter once for all. A pair of dressing forceps with blades closed was introduced from the wound made into the anterior part of the joint to the most dependent part of the outer aspect of the articulation. The instrument was then forcibly thrust through the synovial membrane, the fibrous capsule and the fascia until the point of the forceps was felt under the skin. An incision was then made with a knife through the skin upon the end of the dressing forceps, so as to allow it to protrude. The blades of the forceps were then expanded so as to enlarge the opening which they had made in the deeper structures without risk of causing hæmorrhage. The drain was then seized in the forceps that protruded through the wound and drawn into the joint.

“The ends of the wire were now twisted together and the twisted ends brought out at the wound which was closed with sutures and a small drain inserted. I need hardly say that in this case, as in Dr. Cameron’s, antiseptic treatment was employed.

“It is unnecessary for me to enter into details as to the progress of this case. We have here the temperature-chart for as long as it was thought worth while to have it recorded, and you will see that it indicates, after a little temporary disturbance immediately after the operation, an entirely afebrile condition. The wounds healed without any suppuration. At the end of eight weeks the wire was removed by an incision through the cicatrix. Eight days later the wound made for the removal of the wire had healed. At the end of ten weeks from the operation the patient was allowed to get

up and, though no passive movement had been employed, he could move the limb freely through an angle of about thirty degrees. Two days later he was discharged and unfortunately nothing has been heard of him since. I saw him once in a cart a few days after he was dismissed but I have not been able to learn any further tidings of him. This, I believe, is the first instance of a recent case of fracture of the patella being treated by wire-suture antiseptically applied. My next case occurred two years later."

[From a clinical lecture delivered at King's College Hospital on Dec. 10, 1877. Reported in the *Lancet*. 1878. i. 5.]

A CONTRIBUTION TO THE STUDY OF PULMONARY LOBECTOMY.*

BY A. GRAHAM BRYCE,

ASSISTANT SURGEON, SALFORD ROYAL HOSPITAL; LECTURER IN REGIONAL SURGERY, UNIVERSITY OF MANCHESTER.

THE excision of a lobe of the lung of necessity involves the severance of its connections, normal and abnormal, with the walls of the cavity in which it is confined; and present-day methods of pulmonary lobectomy—the Brunn-Shenstone^{1, 2} and Alexander operations may be taken as types—are dependent, amongst other things, on the feasibility of dividing pleural adhesions when these exist. As a rule, the division is easily effected by dissection with scissors or gauze.†

Not very uncommonly, however, the adhesive pleurisy will be found to have progressed so far that, over a more or less extensive area, the lung and chest wall have become integral with one another, so that no method of separation, other than frank section with a knife, is adequate to effect their division. In these circumstances the freeing of the lung may prove a severe task for the surgeon; it certainly adds considerably to the length of the operation and the burden of the patient, and it entails the risk of wounding the lung and so allowing the escape of highly infective material.

It is suggested that these technically more formidable cases might be dealt with by ligature of the pedicle of the lobe, the gangrenous lung being left *in situ* until it had begun to separate by a line of demarcation between the living and the dead tissues. Before this method can be applied to human surgery it is necessary to estimate the ability of the animal organism to tolerate the presence in the chest cavity of a mass of dead lung.

It was primarily with this object in view that the present investigation was undertaken. The research was carried out in cats and involved three operative stages: (1) To attempt to cause dense pleural adhesions, so that the conditions met with in practice might be imitated; (2) To bring about gangrene of a lobe by ligation of its pedicle; (3) After intervals varying in different animals, to attempt the removal of the dead tissue from within its living surroundings.

* From the Department of Surgery, University of Manchester.

† Alexander's³ technique, which has a bearing on what follows, calls for a short description. At the first stage pleural adhesions are broken down by the finger or divided with scissors. The whole lung and the parietal pleura are then gently rubbed over with gauze. At the second stage the newly-formed adhesions of the diseased lobe are broken down by the finger, the pedicle is doubly ligatured, and, after provision has been made for irrigation and drainage, the lobe is brought out of the wound, which is sutured round the projecting lung. Four days later the wound is opened up completely, and, in due course, the protruding lobe separates, leaving a bronchial fistula.

GENERAL DESCRIPTION OF EXPERIMENTAL METHOD.

The anæsthetic used throughout was ether, with or without $\frac{1}{4}$ mgrm. of atropine. After induction of surgical anæsthesia, a No. 8 English, gum-elastic catheter was introduced into the trachea with the aid of a simple laryngeal spatula and a headlight. Strong traction on the tongue renders exposure of the larynx easy in the cat. The administration of ether was continued by positive ventilation, and the chest was opened through an intercostal incision, the ribs being spread by means of a mastoid retractor.

At the first stage various means of bringing about the desired adhesions were applied, and the opportunity was taken to place a double ligature of ordinary sewing cotton loosely in position round the pedicle of the selected lobe, the ends of the ligature being left under the skin in order to facilitate subsequent identification. At the second stage the ligature was traced to the hilum and there tightly tied so as to deprive the lobe entirely of its blood-supply. Care was taken to reduce the separation of adhesions to the minimum compatible with accessibility of the pedicle. The lobe was left undisturbed and the chest was closed with or without drainage. Reference will be made later to the question of drainage, but it may be said here that, as the experiments went on, the necessity for drainage at any stage appeared less and less. Two animals, Nos. 6 and 7, passed through all three stages without having the chest drained at any of them. In one, No. 5, though it was perfectly well, a considerable serous effusion between the diaphragm and the lower lobe was discovered at the third-stage operation, and in another, No. 6, the meshes of the gauze in which the lobe had been wrapped were soaked in serum. Apart from these and the two fatal empyemata, no effusion was discovered in any animal throughout the course of the experiments.

The Experimental Production of Pleural Adhesions.—In the first two animals friction applied to both layers of the pleura by dry gauze and gauze soaked in rectified spirit failed to engender any but negligible adhesions. In the third and fourth cats an attempt to cause obliteration of the pleural cavity by the instillation of Dakin's solution, which Beck⁴ has used in dogs to produce adhesive pericarditis, resulted in the death of the animals from sero-fibrinous pleurisy and bilateral empyema respectively. Chlorinated solutions were therefore discarded in the subsequent experiments. Finally recourse was had to a method described by Nissen.⁵ He enclosed the lobe in a large-meshed string bag, the neck of which was tied loosely round the pedicle by means of a running noose. In the experiments with which this paper is concerned, Nissen's string bag was at first replaced by a single layer of gauze. This proved bulky and clumsy, and in Cats 7, 8, and 9 the lobe in question was enclosed in a bag of cotton net, made roughly to shape and having a noose to surround the pedicle without compressing it. On the whole, this was a satisfactory method, but the interposition of the gauze or net between the two layers of pleura made the line of demarcation of the living and dead tissues rather different in character from what it would be in human

TABLE SHOWING RESULTS OF EXPERIMENTS.

CAT No.	1ST STAGE	RESULT	INTER-VAL	2ND STAGE	RESULT	INTER-VAL	3RD STAGE	RESULT	REMARKS
1	Left upper lobe. Ligature placed in position round pedicle. Whole of both layers of pleura rubbed over with gauze. No drain	Off food for three days. Wound healed by first intention	Days 31	Ligature tightly tied. 3-day tube drain	Very fit next day. Wound healed by first intention	Days 14	Some adhesions, but much open pleural space. Lobe excised. Tube drain	Very fit next day —walking. The wound healed in 8 days	Developed a 'cold' after complete recovery. Well for over 4 months, then died for no apparent reason. P.M.—Shifting of mediastinum to left. No cause for death found
2	Ligature left loose round left upper lobe pedicle. Whole pleura rubbed over with gauze soaked in spirit. No drain	Wound healed by first intention	15	Ligature tightly tied. Further ligature applied and pedicle cut between them. Practically no adhesions, so that lobe remained freely mobile in pleural cavity	Good recovery for first week, then developed 'pneumonia'. Steady improvement after 14th day. Little pus from wound but quite healed by end of two weeks	35	Gray sloughed lobe firmly adherent to mediastinum and pericardium. Some force necessary to bring about its separation.	Died a few hours after operation	P.M.—Right heart dilated. Little blood in both pleural cavities, though left pleura intact. Right lower lobe collapsed. It and left lung showed considerable areas of deep congestion
3	Ligature left loose round left upper lobe pedicle. Dakin's solution rubbed over whole pleural cavity. Drain, temporarily closed to prevent pneumonia	Recovered from operation, but the animal soon lost ground. Free discharge from wound. Died 10 days after operation							P.M.—Sero-fibrinous pleurisy throughout left chest
4	Pulmonary ligament cut and ligature loosely placed round pedicle of left lower lobe. Diaphragm accidentally injured and sutured. Tube drain temporarily tied at free end. Pl.	Recovered well for 9 days. About 10th day milky discharge from tube. Condition began to deteriorate on 18th day and animal was destroyed on 22nd day							P.M.—Bilateral empyema. Left lung entirely collapsed

6	Pedicle of left lower lobe loosely encircled by ligature. Lobe enclosed in surgical layer of gauze. Tube drain temporarily tied at free end	Recovered well	14	Extensive adhesions. Ligature tightly tied. No drain	Good recovery. (Fig. 375)	14	Separation of lobe with difficulty. Pedicle cut and gangrenous lobe removed. Extensive wound of right pleura. Air-tight closure	Uninterrupted recovery	Animal destroyed 7½ weeks after 3rd stage. Shifting of mediastinum and right lung as in Cat 5 (see Figs. 376, 377)
7	Pedicle of right lower lobe loosely encircled by ligature. Lobe wrapped in net bag, neck being loosely tied round hilum. Lig too small for lobe. No drain	Recovered well	28	Good adhesions, but lobe not everywhere in contact with chest wall. Ligature tightly tied. Healthily lobe injured, but small breach ligatured. No drain	Uninterrupted recovery	7	Adhesions separated fairly easily, but small hole torn in left pleura. Pedicle divided & gangrenous lobe removed. No drain	Uninterrupted recovery	Animal alive and well at time of writing, 17 weeks after 3rd stage
8	Pedicle of left lower lobe loosely encircled by ligature. Lobe wrapped in net bag of ample size. No drain	Good operative recovery, but did not cut well and lost some weight	14	Lobe completely expanded and everywhere adherent. Ligature tightly tied. No drain	Good operative recovery, but animal refused food. Artificial feeding tried, but died on 4th day after 2nd stage				Lower lobe gangrenous & everywhere adherent to upper lobe and chest wall. Specimen shown in Fig. 374
9	Pedicle of right lower lobe loosely encircled by ligature. Lobe wrapped in net bag of ample size. No drain	Uninterrupted recovery	14	Lobe rather collapsed. Adhesions of lower to other lobes, but not to chest wall. Ligature tightly tied. No drain	Good recovery	0	Laceration of healthy lobes during separation and removal of gangrenous lower lobe after division of pedicle. Open drain to prevent tense pneumothorax	Died a few hours after operation	P.M. Little blood in both pleural cavities. Right heart dilated. Both lungs congested

cavity, to have become firmly adherent to the mediastinum and pericardium, so that it could only be separated with great difficulty. In spite of its tough consistence, it tore in two pieces during the process of extraction. The animal died a few hours after the operation. In Cat 9, in which the right lower lobe had been enclosed in a net bag, adhesions had formed only between the devascularized and the neighbouring healthy middle and the upper lobes, and not to the chest wall. In the separation of these adhesions, nine days after the second stage, considerable laceration was caused to the healthy



FIG 375.—Cat 6. Eleven days after second-stage operation. The figure shows the consolidated (gangrenous) left lower lobe, with a small effusion.

lobes. An open tube drain was left in the chest, to prevent any danger of tense pneumothorax. Death took place shortly after the operation. The post-mortem appearances were the same in both animals. Dilatation of the right heart and congestion of both lungs pointed to respiratory failure as the cause of death, though the mechanism of its production was not clear.

The late changes in position of the thoracic contents which occur as the result of lobectomy were seen after death in Cats 1, 5, and 6, and are pictured in the living animal in *Figs. 376, 377*. These changes have been studied by Morrison⁶ and do not call for a detailed description here.

DISCUSSION.

The investigations which form the subject of this paper leave no doubt concerning the possibility of survival of an animal in whose closed pleural cavity there is a necrotic pulmonary lobe. They lend support to the proposal to practise a two-stage lobectomy in man, by depriving the lobe of its blood-supply and later shelling out the gangrenous lobe. It must be borne in mind that the animals used and their lungs had previously been healthy and that



FIG. 376.—Normal cat—for comparison with Fig. 377.

a like success might not attend the unthinking application of the same methods to the abscess-riddled lungs of broken-down human patients.

Recent technical developments in pulmonary lobectomy in man have an important bearing on this question. It is long since Sauerbruch ligatured the main branch of the pulmonary artery to the diseased lobe, but during the past two years there have been published accounts of operations in which the whole pedicle has been constricted, the pulmonary tissue being left to slough inside the chest. Thus Nissen,⁷ after an extensive pneumolysis, removed a whole lung by this method, and he is quoted by Lagos⁸ as using

elastic compression as near the pedicle as possible in those cases in which isolation of the hilum is impracticable. John Alexander,⁹ at the second stage of his routine lobectomy, now ligatures the pedicle and closes the chest tightly round a drainage-tube, re-opening the wound and, in certain cases, cutting away the gangrenous lobe on the fourth or fifth day. In that adhesiolysis is in them an essential preliminary, these operations differ from the one which is here suggested, but the feasibility of the latter in man seems assured by the successes which Alexander and Nissen have reported. Such



FIG. 377.—Cat. 6. Nine weeks after third-stage operation (removal of left lower lobe). The figure shows falling in of left thorax and mediastinal shift to the left.

an operation would not generally replace more orthodox methods of lobectomy, but would find its special indication in cases which may be grouped as: (1) Those in which the firmness of adhesions between the visceral and parietal pleura renders their separation dangerous or impossible; and (2) Those in which previous drainage to the exterior of abscess or bronchiectatic cavities has fused the lung to the chest wall. The projected operation has the advantage of divisibility into multiple stages and is applicable to those cases in which the surgeon considers it desirable to obliterate the pleural

cavity before exseising the lobe: but it would only be practicable where existing accessibility of the hilum or its exposure by dissection permitted isolation and ligation of the pedicle.

SUMMARY.

1. An account is given of experimental work which shows that animals can survive although there is a mass of dead lung in the pleural cavity.

2. The findings support the proposal that it would be possible to remove densely adherent lung by cutting off its blood-supply and allowing it to slough, the extraction of the dead lung being deferred till it had separated by the formation of a line of demarcation between it and its surroundings.

The expenses of this investigation have been met by a grant from the University of Manchester. I desire to express my thanks to Dr. A. D. Macdonald, Reader in Pharmacology in the University, for placing at my disposal the resources of his laboratory, for his continued interest and much personal help; and to Dr. R. S. Paterson, who has taken the X-ray photographs and interpreted them for me.

REFERENCES.

- ¹ BRUNN, HAROLD, *Surg. Gynecol. and Obst.*, 1932, lv, 616.
- ² SHENSTONE, N. S., and JAMES, R. M., *Canad. Med. Assoc. Jour.*, 1932, xxvii, 138.
- ³ ALEXANDER, JOHN, Personal communication, 1931.
- ⁴ BECK, C. S., and GRISWOLD, R. A., *Arch. of Surg.*, 1930, xxi, 1064.
- ⁵ NISSEN, R., *Deut. Zeits. f. Chir.*, 1920, ccix, 389.
- ⁶ MORRISON, J. T., *Brit. Jour. Surg.*, 1926, xiv, 94.
- ⁷ NISSEN, R., *Zentralb. f. Chir.*, 1931, lviii, 3003.
- ⁸ LAGOS, F. M., *Soc. Internat. de Chir.*, 9th Congress. Madrid, 1932.
- ⁹ BALLON, H., SINGER, J. J., and GRAHAM, F. A., *Jour. of Thorac. Surg.*, 1932, i, 533.

MULTIPLE CARCINOMA OF THE COLON: WITH FOUR ORIGINAL CASES.

By A. J. COKKINIS,

A SISTANT DIRECTOR, SURGICAL UNIT, ST. MARY'S HOSPITAL, LONDON.

MULTIPLE PRIMARY MALIGNANCY: ITS FREQUENCY AND CAUSATION.

SINCE Billroth published the first authentic cases of multiple primary cancer in 1869 hundreds of contributions have been made to the literature and several large series of cases have been collected. The subject is of sufficient interest in itself, the occurrence of more than one primary malignant growth in the same patient being always something of a phenomenon. Of greater significance is the possibility that these multiple growths may shed light on some of the unsolved problems of cancer pathology.

Incidence of Multiple Cancer.—The literature reveals a striking lack of agreement on the frequency of this condition. Some writers claim that it is rare and that its occurrence is within the limits of coincidence; others maintain that it is common and that factors other than coincidence are concerned in its etiology. This difference is amply illustrated by *Tables I and II*, which summarize the incidence of multiple primary growths in some of the larger published lists of cancer autopsies. They show a discrepancy too great to be accidental, and accountable for only by variation of the criteria of multiple malignancy employed by the writers.

The fundamental problem confronting every worker in this field is: What exactly constitutes multiple primary malignancy? Should the growths be histologically distinct and heterogeneous? Must they occur in different organs, systems, or tissues? Should only synchronous growths be included, and should a prolonged interval between metachronous growths disqualify them? Must each tumour be associated with distinct and dissociable metastases?

Criteria based on affirmative replies to these questions are necessarily so severe that only a small proportion of possible cases of multiple primary cancer, whether published or unpublished, could survive them. And it is on such or similar criteria that the 'low-incidence' school, following the example set by Billroth, base their statistics. Thus, Orr⁴ agrees with Ewing¹⁰ that there is no etiological relation between multiple malignant tumours, and that their occurrence can be explained by coincidence, while Murray¹¹ goes so far as to state that one malignant growth actually inhibits the development of a second.

A moment's consideration, however, must show that these criteria are too strict, and that observations based on them are unconvincing. Surely, if two malignant tumours coexist, or follow each other, and it can be definitely

established that one is not a metastasis of the other, they must both be regarded as primary growths. And if, on such basis, multiple primary malignancy proves more common than can be accounted for by chance, then the etiological significance of this phenomenon is worthy of investigation.

Table I.—STATISTICS OF 'LOW INCIDENCE' OF MULTIPLE MALIGNANCY IN CANCER AUTOPSIES.

AUTHOR	TOTAL CANCER AUTOPSIES	CASES OF MULTIPLE MALIGNANCY	PERCENTAGE
Junghanns ¹ (1919)	4219	19	0.5
Brandt and Jakobson ² (1930)	2083	11	0.5
Bilello and Montanini ³ (1931)	1154	7	0.6
Orr ⁴ (St. Mary's Hospital) (1930)	1048	8	0.8
Totals	8502	45	0.53

Table II.—STATISTICS OF 'HIGH INCIDENCE' OF MULTIPLE MALIGNANCY IN CANCER AUTOPSIES.

AUTHOR	TOTAL CANCER AUTOPSIES	CASES OF MULTIPLE MALIGNANCY	PERCENTAGE
Hanlon ⁵ (1931)	950	18	1.9
Egli ⁶ (1914)	631	16	2.2
Warren and Gates ⁷ (1932)	1078	40	3.7
Medwedjew ⁸ (1924)	470	20	4.3
Owen ⁹ (1921)*	3000	143	4.8
Totals	6129	237	3.9

* Not strictly an autopsy list.

In this matter I find myself in entire agreement with Warren and Gates,⁷ who include under the term 'multiple primary malignancy' all multiple growths except those with a possible metastatic origin, and those which are commonly pluricentric (e.g., multiple skin cancers, bilateral ovarian tumours, multiple myelomatosis, etc.). On this basis they collected 1259 cases of proved multiple primary malignancy from the literature and from their own records. Their rejection of no fewer than 439 of the 628 cases analysed by Major¹² in 1918 is evidence of the strictness of their criteria. The figures quoted by Warren and Gates show, with some certainty, that the incidence of multiple primary malignancy is many times greater than the calculated incidence based on chance. One may add that probably more cases are missed than incorrectly diagnosed. These considerations lead to the conclusion that multiple malignant growths occur much more frequently than can be accounted for by coincidence.

Etiology of Multiple Cancer.—A study of published cases reveals, as one would expect, that the great bulk are multiple carcinomata, showing all

gradations between exactly similar and totally dissimilar growths; a few are carcinoma and sarcoma in different organs, while the rarest are multiple primary sarcomata. Of peculiar interest are the examples of the carcinoma sarcomatodes of Virchow, in which carcinoma and sarcoma are found in the same organ; Warren and Gates found 71 such cases in the literature. In this connection mention may be made of the experiments of Ehrlich and of Russell (quoted by Norbury¹³), who succeeded in changing typical adenocarcinoma in mice into a mixed tumour, and finally into a pure sarcoma.

Accepting the high incidence of multiple malignancy (probably between 3 and 4 per cent of all cancers), a consideration of its etiology leads us to four possible explanations: (1) Implantation (contact or self-infection); (2) Origin from multiple or diffuse precancerous lesions; (3) Individual susceptibility to malignancy, perhaps inherited or familial; (4) Increased susceptibility (diminished immunity) produced by a previous cancer.

1. *Implantation or contact cancers* are common and well known, but they are not examples of multiple primary malignancy. The possibility of implantation does, however, arise in some not obviously contact cancers, and must be taken into account (*see below*).

2. *Pluricentric origin from precancerous lesions* is undoubtedly responsible for some cases (e.g., malignant polyposis of intestine), but it cannot be sustained as a general explanation. Were this the common etiology, one would expect most multiple growths to attack the same organ or system. Actually, the reverse is the case, multiple cancers being mostly encountered in organs unrelated by any system (Warren and Gates,⁷ Norbury,¹³ Bargen and Rankin¹⁴).

3. *Individual susceptibility* (perhaps hereditary or familial) is the only explanation capable of general application. Accepting this susceptibility it is not difficult to understand why more than one growth develops, provided there is more than one locus of irritation. This view is supported by Slye's experiments (quoted by Norbury¹³), who found that mice with a cancerous ancestry develop carcinoma following injury which has no effect on control mice of non-cancerous ancestry.

4. Whether or not one cancer produces *increased susceptibility* to a second we have no means of determining. Cuthbert Dukes,¹⁵ writing in 1930, considers that existing statistics fail to settle the question either way. But more recent publications, especially Warren and Gates' painstaking analysis, appear to establish (one hopes beyond dispute) the cardinal fact that *one primary malignant tumour does not inhibit the development of a second*. In other words, that cancer does not confer immunity.

MULTIPLE CARCINOMA OF THE COLON.

It is somewhat surprising to find that while multiple cancer of the gastro-intestinal tract is far from rare (169 cases were collected by Warren and Gates), apparently only 29 authentic cases are reported of multiple growths limited to the colon. In most of the published cases the rectum, stomach, and small gut, in that order of frequency, share the growths with the colon, or harbour them to its exclusion. The occurrence of 4 cases

of multiple colonic growths, out of a total of 54 cases of cancer of the colon attended in the Surgical Unit of this Hospital in the past ten years, must therefore be a unique experience.

A brief description of the cases (hitherto unpublished) is here given:—

Case 1.—Carcinoma of descending colon. Carcinoma of pelvic colon.

Female, aged 66 (1929). Occasional diarrhoea and melena for three and a half years. Radiograms show stasis in the sigmoid colon. Operation revealed an annular malignant stricture at lower end of pelvic colon, and a second distinct carcinoma of descending colon. Inoperable owing to secondary growths along mesentery. Ovarian cyst also present. Transverse colostomy performed. Patient discharged relieved.

Comment.—The long history, and the presence of peritoneal secondaries, and of an ovarian cyst, are noteworthy. Owing to the absence of any indication of the relative ages of the two growths, the possibility of implantation cannot be excluded.

Case 2.—Four separate cancers of left colon (*Fig. 378*).

Male, aged 64 (1922). Pain, constipation, and blood in stools (eleven months). Lump felt along iliac colon and another in rectovesical pouch. Operation revealed



FIG. 378.—*Case 2.* Descending and sigmoid colon, showing four carcinomatous growths. (*St. Mary's Hospital Museum.*)

large growth of pelvic colon, and three smaller growths more proximally (one in iliac colon, two in descending colon). Resection of left colon and anastomosis. Uneventful recovery.

PATHOLOGICAL REPORT.—Examination shows four independent polypoid growths of papillary, alveolar, tubular and trabecular, columnar and spheroidal-celled adenocarcinoma. At least one benign adenomatous polyp is present.

Comment.—This is clearly an example of pluricentric malignant change in a multiple adenomatosis of the colon. The more advanced stage of the distal growth diminishes the likelihood of implantation.

Case 3.—Carcinoma of pelvic colon. Carcinoma of transverse colon.

Female, aged 68 (1922). Four months previously cæcostomy (? blind) performed for obstruction. Now obstruction recurred owing to narrowing of cæcostomy. Radiograms show obstruction at junction of iliac and pelvic colon. Operation discovered a fixed malignant mass involving pelvic colon. Left iliac colostomy

attempted, but sigmoid could not be delivered. In attempting transverse colostomy, a second and ring-like growth was discovered in its middle. This was more recent and removable; it was resected and a transverse colostomy established. Patient relieved.

PATHOLOGICAL REPORT.—Ulcerated, alveolar and tubular, columnar-celled, scirrhous carcinoma.

Comment.—The accidental discovery of the second growth in the performance of a colostomy is the outstanding feature. The more advanced condition of the distal growth excludes the chance of implantation, and there is nothing to suggest an origin from preceeding adenomatosis.

Case 4.—Carcinoma of transverse colon. Carcinoma of pelvi-rectal junction. (Author's case.)

Male, aged 60 (1932). Two months' pain, constipation, and diarrhoea. Radiograms show filling defect in transverse colon and abnormal appearance in lower sigmoid. At operation, freely movable, ring-like growth of transverse colon found. Exploration of pelvis for secondaries revealed a second ring-like growth at pelvi-rectal junction; this was larger and somewhat fixed by recent adhesions. Both the growths resected. Temporary transverse colostomy established at proximal resection and intussusception-anastomosis performed at distal resection. Patient discharged fit, but died from liver metastases nine months later.

Comment.—As in *Case 3*, the second growth was discovered during the exploration necessitated by the first growth. Here, again, the distal growth is the older of the two.

DISCUSSION.

The occurrence of these four cases of multiple cancer of the colon in one surgical firm of this hospital in a period of ten years is surprising in view of the very small number of cases in the literature. Norbury,¹³ in his Presidential Address at the Royal Society of Medicine (1930), was able to report only two cases, and to collect three more from the records of three of the larger London hospitals. Loekhart-Mummery,¹⁶ in the discussion which followed, reported five cases of multiple cancer of the alimentary tract, but in none of these was there more than one colonic growth. Abel¹⁷ in 1929 showed three specimens of multiple cancer of the intestine, but only one of these had more than one growth in the colon. Maingot's¹⁸ case presented one growth in the anus, one in the rectum, and only one in the colon.

Bargen and Rankin¹⁴ contribute the largest group of cases in the literature. They mention a case described by Fenger (1888) in which a transverse colostomy was done for a growth at the splenic flexure, with no relief; at autopsy a second growth was found in the ascending colon. They also refer to a case reported by Dowden (1917) in which four metachronous cancers of the colon developed in six years. Finally, they contribute sixteen cases of proved multiple cancer of the intestine, in six of which there were two or more growths in the colon. Three of these six cases are obvious examples of carcinoma supervening on multiple adenomatosis. Case 6 is of great interest: a cancer of the ascending colon was resected in 1920; in 1926 a carcinoma of the sigmoid was resected; in 1929 a third growth was found in the splenic flexure and removed, while in the same year a hysterectomy was done for sarcoma of the uterus!

The accidental discovery of the second growth in two of my cases (*Cases 3 and 4*), and in several cases in the literature, suggests that multiple growths of the colon would be found more frequently if a thorough search were made for them in every case that comes to operation or post-mortem. The clinical importance of such a search is obvious. To overlook a second growth during a resection for operable carcinoma would be a major catastrophe, while colostomy has been performed more than once for inoperable cancer, only to prove a complete failure because a second growth existed higher up. Finally, the possibility of a metachronous growth developing at some future period must influence the prognosis of all operable cancers of the rectum or colon.

Etiology of Multiple Cancer of the Intestine.—When two or more cancers are found in the intestine the first and most obvious explanation is that one is the primary growth and that the others are implantation secondaries. Unfortunately this argument is inadmissible in the great majority of recorded cases, because either the most distal growth is the oldest (e.g., *Cases 3 and 4* of this series), or the growths are too far apart, or the interval between metachronous growths is too long. Nevertheless, implantation is a possible cause in a few cases (Maingot's case and *Case 1* of this series).

Pre-existent adenomatous polyposis is a very definite etiological factor. In several published cases (*Case 2* of this series, *Cases 3, 13, and 15* of *Bargen and Rankin's* series, and others) there is unmistakable evidence, both clinical and pathological, of malignant transformation of preceding adenomata. The majority, however, present no such evidence, and can only be attributed to individual susceptibility, negative immunity, or, of course, coincidence.

By inference, one might argue that a similar etiology operates in single cancers of the colon, except that precancerous adenomatosis is unlikely to play quite as prominent a part.

The association between adenomatous polyposis and carcinoma of the rectum and colon is of obvious importance. Loekhart-Mummery and Cuthbert Dukes¹⁹ consider that this association is frequent, and describe three stages in the transformation: (1) *Stage of hyperplasia*, with irregular epithelial proliferation over extensive areas of the gut; (2) *Stage of polyposis*, with visible sessile and multiple adenomatous polypi; (3) *Stage of malignancy*, one or more of the polypi becoming carcinomatous. They allege that with the development of carcinoma the benign adenomata and hyperplasias retrogress and disappear. If this is true, it would seem that precancerous adenoma might be a common forerunner of cancer of the intestine; in the absence of actual proof, however, this retrogression appears to be somewhat fanciful. Nevertheless, it cannot be denied that there are specimens showing benign adenomata and adenomata undergoing malignant change side by side, and that carcinoma is a potential danger in intestinal polypi.

Actually, three distinct forms of intestinal polypi can be recognized: (1) A familial disorder of young adults, perhaps traceable through several generations, to which the term 'polyposis intestini' has been applied, and which, according to Doering,²⁰ carries a 60 per cent risk of ultimate carcinoma; (2) The more common and less defined condition known as 'adenomatosis', similar to the above, but without a definite familial or age incidence; (3) The 'pseudo-adenomatous' polypoid hypertrophies and hyperplasias which are seen

in chronic inflammatory lesions of the gut, such as tubercle and bilharziasis. In the present state of our knowledge it is safest to assume that all these types of intestinal polyposis are potentially precancerous, and to act accordingly.

CONCLUSIONS.

1. Multiple primary malignant growths occur more frequently than can be accounted for by coincidence.

2. In some part this may be attributed to the presence of multiple or diffuse precancerous lesions, but a more likely general explanation is an individual susceptibility to malignant disease.

3. One growth does not confer immunity against a second.

4. Multiple carcinoma of the intestine has the same etiology as multiple primary growths elsewhere.

5. Multiple adenoma is a definite cause of multiple cancer of the colon and rectum.

6. All forms of intestinal polypi are potentially malignant and should be treated as such.

7. The possibility that more than one growth may be present or develop should be remembered at all operations for cancer of the intestine, and also in the prognosis.

REFERENCES.

- ¹ JUNGHANNS, *Zeits. f. Krebsforsch.*, 1929, xxix, 623.
- ² BRANDT and JEKABSON, *Ibid.*, 1930, xxxii, 280.
- ³ BILELLO and MONTANINI, *Tumori*, 1931, xvii, 369.
- ⁴ ORR, *Jour. Pathol. and Bacteriol.*, 1930, xxxiii, 283.
- ⁵ HANLON, *Amer. Jour. Cancer*, 1931, xv, 2001.
- ⁶ EGLI, *Corr.-Blatt. f. schweiz. Aerzte*, 1914, lxiv, 449.
- ⁷ WARREN and GATES, *Amer. Jour. Cancer*, 1932, xvi, 1358.
- ⁸ MEDWEDJEW, Quoted by Warren and Gates.
- ⁹ OWEN, *Jour. Amer. Med. Assoc.*, 1921, lxxvi, 1329.
- ¹⁰ EWING, *Neoplastic Diseases*, 1928 (quoted by Orr).
- ¹¹ MURRAY, *Lancet*, 1927, ii, 800.
- ¹² MAJOR, *Bull. Johns Hopkins Hosp.*, 1918, xxix, 223.
- ¹³ NORBURY, *Proc. Roy. Soc. Med.*, 1931, xxiv, 198.
- ¹⁴ BARGEN and RANKIN, *Ann. of Surg.*, 1930, xci, 583.
- ¹⁵ DUKES, *Cancer Review*, 1930, v, 570.
- ¹⁶ LOCKHART-MUMMERY, *Proc. Roy. Soc. Med.*, 1931, xxiv, 206.
- ¹⁷ ABEL, *Ibid.*, 1929, xxii, 1330.
- ¹⁸ MAINGOT, *Trans. Med. Soc. Lond.*, 1927, i, 121.
- ¹⁹ LOCKHART-MUMMERY and DUKES, *Surg. Gynecol. and Obst.*, 1928, xlv, 591.
- ²⁰ DOERING, *Arch. f. klin. Chir.*, 1907, lxxxviii, 194.

SOME EXPERIMENTAL OBSERVATIONS BEARING ON THE ETIOLOGY OF MEGACOLON.

BY HAROLD BURROWS, C.B.E.,

EXPERIMENTAL PATHOLOGIST, RESEARCH INSTITUTE OF THE CANCER HOSPITAL (FREE), LONDON.

IN the course of an inquiry at the Research Institute of the Cancer Hospital (Free), London, into the part played by irritation in the causation of cancer, the following experiment was performed on eleven young rats: Some powdered crystalline silica was introduced into the mesentery of the cæcum, and a fortnight later intraperitoneal injections of 1:2:5:6-dibenzanthracene were given and thereafter repeated once a week, 1 c.c. of a 0.1 per cent solution of the hydrocarbon in emulsified olive oil being used for each dose. The object was to learn whether a fixation of the carcinogenic agent would result from the chronic inflammation induced by the silica and so cause a tumour to appear in its vicinity.

In one rat only of this series (Rat 8 in *Fig. 379*) did a tumour arise. This rat was killed on account of ascites fifty-three weeks after the injection

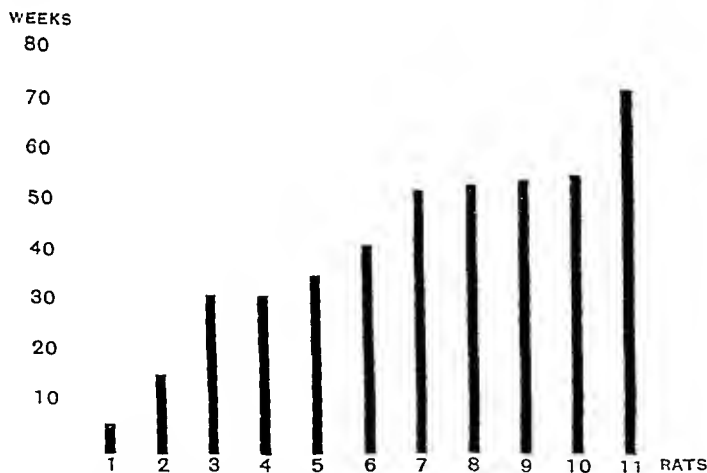


FIG. 379.—The vertical lines refer to the duration of life, in weeks, of each rat after the injection of silica into the mesentery of the cæcum. The numbers 1 to 11 at the foot of the vertical lines represent the individual rats. The numbers 10 to 80 at the left-hand side of the figure indicate weeks. All the rats had enlargement of the cæcum at death.

of silica, and was found to have a large solitary spindle-celled tumour in the anterior part of the abdomen, invading the spleen and pancreas. The cæcum and its mesentery were free from growth.

A remarkable feature observed in every one of the eleven rats treated in this way was an enlargement of the cæcum (*Figs. 380-383*). Although this increase in volume of the gut was extensive enough in some of the animals to be detected during life, they appeared to remain in good health.



FIG. 380.—Rat 3, killed thirty-two weeks after the injection of powdered crystalline silica into the mesentery of the cæcum.

The rat with the most pronounced enlargement of the cæcum (No. 5) died thirty-six weeks after the beginning of the experiment and putrefaction prevented a good illustration. The smallest cæcum in this series was found in Rat 9 (*Fig. 383,A*), and in this case the viscus was fixed to the neighbouring structures by extensive adhesions.

Fig. 379 shows the duration of life in weeks of each animal after the introduction of silica into the mesentery of the cæcum.

DISCUSSION.

Admittedly the histological and anatomical study of the cæcal enlargements in these rats has been incomplete, and no control experiments were made. For these reasons the experiment is being repeated. Meanwhile it

FIG. 381.—A, Cæcum of Rat No. 7, fifty-two weeks after the injection of silica into the mesentery of the cæcum; E, Normal rat with cæcum exposed for comparison.



FIG. 382.—Rat 4, killed thirty-two weeks after the injection of silica into the mesentery of the cæcum.

may be justifiable to consider the cause of the enlargements. The first suggestion is that they might have been the consequence of some obstruction of the bowel either by adhesions or by a torsion of the mesentery. No clear evidence is visible of such blockage, and the rats did not appear to suffer from ill health, nor did they eventually die from intestinal obstruction.

The most plausible explanation seems to be that the enlargement has resulted from an overaction of the sympathetic nerves as a consequence of the continued irritation produced by the powdered silica—an irritation to



FIG. 383.—A, Caecum of Rat 9, fifty-six weeks after the injection of silica into the mesentery; B, Rat 6, forty weeks after the injection of silica.

which the parasympathetic nerve-supply presumably would not be exposed owing to its different anatomical relationships. The function of the sympathetic nerves is said to be a stimulation of the circular muscle fibres of the sphincters; while this stimulation continues the action of the parasympathetic nerves, which supply the other muscle fibres of the gut, is inhibited. The modern operation of ramisectomy and sympathectomy as introduced by Wade and Royle¹ for the cure of megacolon puts the responsible overactive sympathetic nerves out of action, leaving the parasympathetic system intact.

Powdered crystalline silica is a persistent irritant when lying in the tissues : and if the current hypothesis which attributes megacolon to overaction of the sympathetic nerve-supply of the segment of bowel concerned is true, then it would not be surprising if the condition could be produced in animals experimentally by causing chronic irritation of the appropriate sympathetic nerves.

The cæcal enlargement in rats reported in the present paper followed quickly after the injection of silica, and in one animal (No. 5) was observed during life, although not especially sought or expected, within fourteen days.

SUMMARY.

Enlargement of the cæcum following injections of crystalline silica into the mesentery of the cæcum is recorded as occurring in rats. A possible bearing of this observation on the etiology of megacolon is suggested.

REFERENCE.

- ¹ WADE, R. B., and ROYLE, N. D., *Med. Jour. of Australia*, 1927, i. 137.

THE PATHOLOGY OF ACUTE STRANGULATION OF THE INTESTINE.

By ROBERT L. HOLT,

ASSISTANT SURGEON, CHRISTIE HOSPITAL, MANCHESTER.

INTRODUCTION.

It is rather surprising that such a common clinical condition as acute strangulation should have received so little attention from the innumerable workers who have attempted to solve the complicated problem provided by the group of conditions which fall under the general heading of acute intestinal obstruction. Acute strangulation has long been differentiated clinically from simple intestinal obstruction, and, whilst this difference has been recognized by many experimental workers,¹⁻⁷ only a few of them have dealt with experimentally produced strangulation. The cause of death has usually been considered to be a toxæmia resulting from the absorption of toxic substances produced in the strangulated segment of bowel. This view was derived from the bulk of experimental evidence then available, for it had been shown that the intestinal content of a strangulated loop when injected, preferably intravenously, into another animal caused the rapid death of that animal. Among the earlier workers Von Albeck⁸ considered that death was due to the absorption of putrefactive toxins formed in the strangulated loop, whilst Murphy and Vincent⁴ believed that the symptoms and death were due to a 'bacterial endotoxæmia'. Eisberg⁹ was one of the few workers who did not completely accept the general view of the cause of death, and suggested that death sometimes took place long before there had been time for bacterial action to play a part in the result. All were agreed that strangulation was a more acute condition than simple obstruction and that death took place more rapidly in the former condition.

The blood chemistry of acute strangulation has been studied by many workers, and their findings are in general agreement. Death takes place so rapidly that dehydration from vomiting does not occur and the changes in the chemistry of the blood and urine are not marked. Copher and Brooks¹¹ have reported death in acute strangulation without blood changes, but there is usually a rise in the blood-urea. Foster and Hausler¹⁰ concluded from their investigations that whilst dehydration, hypochloræmia, and alkalosis were not present to any appreciable degree there was a moderate increase in the urea and non-protein nitrogen. This increase was evidently due to retention, since nearly all animals dying from strangulation have an almost complete anuria. As might be expected from such blood findings, the administration of sodium chloride solutions does little good in acute strangulation.

The term 'strangulation' implies an interference with the blood-supply, and it is evident that this interference may affect the arteries alone, the veins

alone, or both arteries and veins simultaneously. Only the latter two conditions require consideration in strangulation obstruction where the mechanism which causes an obstruction of the bowel also affects the blood-supply.

Vinecent and Murphy⁴ first called attention to the early onset of intoxication in cases of acute obstruction accompanied by a venous obstruction. But it was not until 1924, when Foster and Hausler¹⁰ published their work on acute strangulation, that further light was thrown on this subject. These two workers carried out a most interesting series of experiments. They first tied off varying lengths of bowel in such a way as to cause a complete venous obstruction, and records of the temperature, pulse-rate, respiration-rate, and blood-pressure were taken at definite intervals. They found that their results varied with the length of bowel affected. In cases with long segments the blood-pressure fell rapidly, the pulse-rate increased very markedly, the temperature fell, and the animal died from shock before toxæmia could develop. With the shorter segments the fall in blood-pressure was only moderate, the pulse-rate was not extremely rapid, and the animals overcame the primary shock only to succumb later to a terrific toxæmia and peritonitis. The differences between long and short loops were brought out more clearly when they repeated the above experiments enclosing the strangulated loop in a thin rubber bag which prevented the absorption of toxins via the peritoneum from the strangulated segments. They found that in the long-segment group the use of a rubber bag did not affect the outcome, whilst in the short-segment group the length of life was markedly lengthened. Later experiments were carried out in which the intestine was tied so tightly that a complete and instantaneous anæmia was produced. This group presented an entirely different picture. There were none of the symptoms of acute collapse so evident in the venous obstruction series. Instead they found a slight gradual rise in the pulse-rate and a practically normal blood-pressure during the first fifteen hours. The onset of peritonitis was then indicated by a rapid rise in the temperature and respiration-rate.

It was evident from these experiments that the cause of death in cases of acute strangulation in which venous obstruction alone exists differs from similar cases in which both arteries and veins were occluded. It is possible, therefore, to divide cases of experimental strangulation into two groups: (1) Those cases in which venous obstruction alone is produced; (2) Those cases in which both arteries and veins are tied. This grouping will be used in this paper to simplify the account of the experiments performed by the writer. It is also preferable to subdivide the venous-obstruction group according to the length of bowel strangulated. Foster and Hausler,¹⁰ in commenting on their results, stated that: (1) In long loops the predominant lethal factor is shock; and (2) In shorter loops shock, although present, is not of sufficient intensity to cause death, and time is allowed for toxæmia and peritonitis to develop.

OBJECTS OF THIS PAPER.

It appeared impossible to the writer to obtain a clear and detailed explanation of the pathology of acute strangulation in spite of the excellent experimental work already published. After a careful study of the literature

the following problems were selected for solution : (1) The cause of the circulatory collapse in venous strangulation of long loops of intestine ; (2) Is the absorption of toxic substances from a strangulated loop the cause of death when the loop is only of moderate length ? (3) What is the route of absorption of such toxins and their mode of production ? (4) An explanation of the cause of death when very short loops of gut are strangulated.

The experiments reported in this paper were carried out in an effort to clear up these remaining problems. In all cases dogs were used for the experimental work and every effort was made to prevent any unnecessary pain or discomfort to the animals. All abdominal operations were done under ether anaesthesia preceded by an injection of morphia, and a full aseptic technique was maintained throughout. The blood-pressure readings were carried out by means of a small cannula inserted into the femoral artery through an incision made at the time of the abdominal operation, and it was found that repeated readings could be obtained without loss of blood or discomfort to the animal. At the outset it was realized that, in order to obtain reliable results, the strangulation of the loop must be carefully produced. The encircling ligature used by many of the earlier workers was not employed. The closed loop was produced by using separate tape ligatures obstructing the length of bowel required, and strangulation was produced by dissecting out the vessels in the mesentery and tying them separately with fine silk ligatures.

THE PATHOLOGY OF ACUTE STRANGULATION OF THE INTESTINE IN WHICH VENOUS OBSTRUCTION ALONE EXISTS.

The results of the work already published on this problem may be summarized briefly as follows : Death in acute strangulation is due to a varying combination of shock and toxæmia, and the preponderance of one or other of these factors is determined by the length of bowel involved and the degree of venous occlusion produced. For the purposes of this paper the experiments and results are divided into three groups according to the length of bowel involved in the strangulation. The groups are : (I) *Strangulation of long loops of intestine, 40 cm. or more ;* (II) *Strangulation of moderate-sized loops, 20 to 40 cm. ;* (III) *Strangulation of short loops, less than 20 cm.* This subdivision has been adopted because the results suggest that a different factor is the main cause of death in each of the three groups.

I. STRANGULATION OF LONG LOOPS OF SMALL INTESTINE

The experiments of Foster and Hausler¹⁰ have already been mentioned in which they charted the changes in the blood-pressure, pulse-rate, etc., which take place in animals with strangulation of long loops of intestine. When their results were charted it was found that a rapid and steady fall in the systolic blood-pressure from an average of 150 mm. of mercury to 40 mm., and a rise in the pulse-rate from about 80 or 90 beats per minute to about 240, took place and that death occurred soon after these figures were reached. They concluded that shock was the main lethal factor in these cases and suggested that the shock was due to a combination of the pain resulting from

the twisting of the mesentery, the inactivity of the ductless glands, and the hæmorrhage into the loop. The first object of the experimental work to be reported was an investigation into the exact cause of the shock which is so marked a feature in these cases.

1. **The Cause of the Circulatory Collapse Associated with Strangulation of Long Loops of Intestine.**—It is not the intention of the writer in this paper to enter into a discussion on the merits of the numerous explanations of the circulatory collapse which is commonly known as shock. The work of Blalock and his associates,¹² showing that the symptoms of collapse in traumatic shock are due to the withdrawal of large quantities of blood or blood-plasma from the circulation into the traumatized area, provides the most reasonable theory so far produced. They studied the effects of the rapid loss of blood and reported that recovery was problematical if the loss exceeded an amount equal to 50 per cent of the blood volume or 4 per cent of the body weight. The significance of these results is readily appreciated when the changes taking place in an obstructed loop of intestine, after the ligation of its veins, are considered. The course of events in such a strangulated loop has been well described by McIver and White.¹³ They reported the changes as follows: "The colour of the strangulated loop changes immediately to a dusky red, soon turns a dark red, and finally becomes black. At the same time intense tonic contractions of the whole loop or isolated segments take place and these contractions continue for several hours. Distension of the loop takes place shortly after the obstruction is produced and the thick red exudate found in the lumen is composed largely of blood cells and serum. The peritoneal coat remains smooth and glistening and only in those cases where strangulation has existed about twenty-four hours is a thin coating of fibrin found on the surface. The loop increases in length sometimes as much as three times its original length, and the corresponding changes take place in its diameter. Sections of the bowel wall show a considerable disintegration of the normal structure, the tissue spaces are filled with red blood cells and the mucosa is almost completely degenerated." The appearance of such a loop suggests that it contains an excessive quantity of blood which is being withheld from the circulation. The following experiment from the author's series illustrates the circulatory changes taking place.

Experiment I.—Adult dog. Weight $13\frac{1}{2}$ kilos.

11.0 a.m. $\frac{1}{4}$ gr. morphine.

11.30 a.m. Operation. Ether anæsthesia. A loop of small intestine about 100 cm. in length obstructed by tape ligatures. The veins draining the loop were dissected out in the mesentery and ligated with fine silk. The anastomotic channel along the mesenteric border of the intestine was also ligated with silk.

	B.P.	P.
	150	90
12.30 p.m.	135	135
1.30 p.m.	110	170
3.10 p.m.	45	240

When the last reading was made the dog was unconscious and moribund. Death occurred shortly afterwards and an immediate autopsy was performed. The strangulated loop was found unperforated, very distended, and its walls much

thickened. The loop was removed intact and weighed. It was estimated to have gained in weight the astonishing amount of 570 grm., which was equivalent to 4.5 per cent of the body weight or to more than 50 per cent of the blood volume.

Further experiments showed that loops of intestine treated in the same manner were capable of gaining weight in the proportion of 125 to 150 grm. per 25 cm. length. When the gross and microscopic appearance of a strangulated loop is considered it is at once evident that this gain in weight represents the amount of blood which has been withdrawn from the circulation. As corroborative evidence McIver and White¹³ reported a considerable decrease in the blood volume of cats dying from acute strangulation. In the above experiment more than 50 per cent of the total blood volume was removed from the circulation in the space of about four hours by the strangulated loop, and the blood-pressure and pulse readings testify to its effect on the circulation. It is evident, therefore, that in venous strangulation of long loops of intestine (40 cm. or more in a dog of average weight) blood may be rapidly withheld from the circulation in sufficient quantity to cause death.

2. The Relation of Toxæmia to the Cause of Death in Strangulation of Long Loops.—The theory that death in acute strangulation is always associated with the absorption of toxic substances from the strangulated loop is a view which is still widely held. Eisberg⁹ was one of the earlier workers on the subject who suggested that death sometimes occurred so rapidly that it was doubtful whether there had been time for bacterial action to produce toxins in sufficient quantity to cause death. The first direct evidence on the subject was produced by Foster and Hausler.¹⁰ They enclosed the strangulated loop in a rubber bag in such a way that absorption from the loop by the peritoneal serosa was prevented without interfering with the distension of the segment by blood and gas. When long loops were strangulated their experiments showed that life was not prolonged by this procedure. Furthermore, they were unable to demonstrate that the fluids which accumulate in the peritoneal cavity or in the lumen of a strangulated loop of less than twelve hours' duration possess any marked toxic properties when injected intraperitoneally into other dogs, even in massive doses. The results were confirmed by the writer. Whilst repeating these experiments of Foster's large quantities of the fluid exuding from such long loops were collected in rubber bags by the writer. Intravenous injection of large quantities of this exudate (as much as 200 c.c. in one dog) produced nothing more serious than a transient weakness and tremor of the limbs accompanied by salivation and vomiting. (It is interesting to note that intraperitoneal injection of this fluid into guinea-pigs frequently produced death with all the symptoms of histamine poisoning. This illustrates well the danger of utilizing different species of animals for such injection experiments.) It is evident from these experiments that toxæmia is of little if any importance in causing the death of animals with strangulation of long loops of intestine.

3. Conclusion.—Evidence has been brought forward to show that the symptoms of circulatory collapse associated with an acute strangulation of a long loop of intestine is due to the rapid withdrawal of a large amount of blood from the circulation by the loop—a conclusion also reached by Scott and Wangenstein.¹⁵ Toxæmia has been shown to be of minor importance

in this group of cases, and, in the author's opinion, the cause of death is the acute reduction of the circulating blood volume.

II. STRANGULATION OF LOOPS OF MODERATE LENGTH.

1. **Circulatory Changes.**—The withdrawal of blood from the circulation by a strangulated loop of intestine does not continue indefinitely. The loss is most rapid in the first few hours after strangulation is produced, but is then progressively limited by the thrombosis which, beginning in the veins and capillaries, finally spreads to the smaller arteries. It is evident, therefore, that medium and short loops when strangulated are unable to cause death in the same manner as long loops of gut. Foster and Hausler¹⁰ added considerably to the solution of this problem. They studied the circulatory changes taking place when medium-sized lengths of gut (15 to 25 cm.) were strangulated. They found that the blood-pressure fell in the first six hours to approximately 110 mm. systolic and remained at this level until several hours before death. This stationary period was followed by a very rapid fall, often amounting to 60 mm. in an hour's time. The pulse-rate showed a corresponding rise within the first six hours to an average of 140 beats per minute. After about sixteen hours the rate rose to about 200 and continued at or about that level until death. The following experiments from the writer's series are good illustrations of the circulatory changes :—

Experiment II.—Bitch. Weight 10 kilos.

3.30 p.m. B.P. 145. P. 90.

Operation. $\frac{1}{4}$ gr. morphine and ether anæsthesia. A 25-cm. loop of jejunum obstructed with tape ligatures. Veins draining the loop dissected in mesentery and tied with silk ligatures.

	B.P.	P.
11.0 p.m.	110	160
11.30 a.m.	105	220
2.0 p.m.	105	220
5.0 p.m.	100	240
7.0 p.m.	Dead.	

An immediate autopsy showed the loop distended and not perforated. There was a fair quantity of dark fluid in the peritoneum, but no macroscopic evidence of peritonitis.

Experiment III.—Bitch. Weight 9 kilos.

2.30 p.m. Operation. $\frac{1}{4}$ gr. morphine and ether anæsthesia. Loop of small intestine 25 cm. long, about 45 cm. below Treitz ligament, obstructed and veins ligated. At conclusion :—

	B.P.	P.
	135	90
9.30 p.m.	120	170
11.30 a.m.	120	190
1.30 p.m.	115	240
4.30 p.m.	60	300
7.0 p.m.	Dead. Autopsy showed loop intact.	

The initial fall in blood-pressure is easily explained by the loss of blood into the loop, and the stationary period corresponds to the stage when

thrombosis has become well marked. The terminal fall in pressure requires explanation. Foster and Hausler¹⁰ repeated these experiments, enclosing the loop in a rubber bag. They found that they were able to prolong the lives of the animals for several days. They therefore concluded that the terminal fall in blood-pressure was due to the onset of toxæmia and peritonitis. Chenut,¹⁴ in his experiments, showed that life could be prolonged in cases of strangulation if the strangulated loop were sutured outside the abdominal cavity. He found that the loop sloughed away and the animals died of the resulting fistula. The following experiment from the author's series is quite in keeping with Chenut's results.

Experiment IV.—This was a repetition of Foster and Hausler's experiment of enclosing the strangulated loop in a rubber bag with the addition that the rubber bag was drained on to the surface of the body. This was found to be necessary because in a few earlier experiments fluid collected in the rubber bag in such quantity as to force its way past the ligature holding the bag over the loop. Previously a lateral anastomosis between the bowel proximal and distal to the loop to be strangulated was performed.

Feb. 5, 1932.—Morphine and ether anæsthesia. Preliminary lateral anastomosis performed.

March 2.— $\frac{1}{4}$ gr. morphine half an hour before operation. Ether anæsthesia. A 20-cm. loop of small intestine obstructed and its veins ligated. The loop was enclosed in a rubber bag and sutured extraperitoneally. Later in the day 40 c.c. blood-tinged, odourless fluid removed from the bag.

March 3.—Animal well and walking around cage; 80 c.c. fluid, almost black, light foul odour, removed from rubber bag. Tube stitched into rubber bag draining on the surface of the abdominal wall.

March 9.—Dog perfectly well. Utilized later for other experiment.

The lateral anastomosis was performed so that the picture should not be complicated by a small-gut obstruction, which in itself is liable to be fatal in an experiment of more than a few days' duration. This dog was perfectly well eight days after the experiment. It is evident that life in these cases is prolonged if absorption of substances from the strangulated loop is prevented. It appears, therefore, that the terminal fall of blood-pressure which occurs in the strangulation of intestinal loops of moderate length is due to the absorption of substances from the loop.

2. Evidence to Show that Toxic Substances are Present in the Fluid Exuding from a Strangulated Loop.—Absorption of substances from a strangulated loop is possible by two paths, the lymphatic channels or the peritoneal cavity. It has been found by experiment that the result is not altered by ligating the lymphatics in the mesentery, and it is generally conceded that absorption takes place via the peritoneal cavity. Whilst there is no doubt of the toxicity of the loop contents of these dogs dying of intestinal strangulation, all attempts to prove the presence of a toxin in the peritoneal fluid in such cases have been unsuccessful. Scott and Wangenstein¹⁶ concluded that the peritoneal fluid obtained from dogs dying of acute strangulation is non-toxic when injected intravenously into normal dogs, providing the strangulated loop is not ruptured or about to rupture and the fluid does not give off a foul odour. Wilkie⁷ obtained similar results, but added that "these negative findings only prove that there is insufficient toxin in the

peritoneal fluid at the time of death and does not preclude a steady continuous absorption. the cumulative effect of which is sufficient to produce toxæmia and death". That toxic substances are present in the exudate from a strangulated loop is proved by the following experiment of the writer's:—

Experiment V.—Bitch. Weight 10 kilos.

2.30 p.m. Operation. Loop of jejunum 25 cm. long and about 30 cm. below Treitz ligament obstructed. Loop enclosed in rubber bag draining on to the surface of abdomen. At conclusion of experiment:—

B.P. 125. P. 160.

11.20 a.m. B.P. 120. P. 180.

Contents of rubber bag removed and re-injected intraperitoneally.

1.0 p.m. B.P. 105. P. 200.

2.30 p.m. B.P. 30. P. not countable.

2.35 p.m. Dead. Immediate autopsy. Loop had not perforated.

In this experiment all the fluid exuding from outside the loop during the first twenty-one hours of strangulation was collected by the rubber bag and was not diluted by the addition of any peritoneal fluid. It was injected intraperitoneally to utilize as far as possible the normal route of absorption. Its effect is shown by the blood-pressure and pulse records, death occurring about three hours after the injection. An immediate autopsy was performed and the strangulated loop was found to be intact so that it was clear that the exudate had not been contaminated by any substances resulting from a rupture of the loop. It has, however, been shown that the fluid exuding from strangulated loops within the first twelve hours after onset is non-toxic.¹⁰ Experiments were carried out by the writer in order to find out if possible the time at which the exudate begins to contain toxic substances. The following is a typical example of the results obtained:—

Experiment VI.—Dog. A loop of jejunum 25 cm. in length obstructed and its veins ligated. Loop enclosed in a rubber bag into which a tube was inserted draining to surface of abdomen.

2.0 p.m. Operation. $\frac{1}{4}$ gr. morphine and ether anaesthesia.

4.30 p.m. B.P. 120. P. 140.

10.30 p.m. B.P. 100. P. 160.

90 c.c. fluid, odourless, slightly blood-stained, coagulating on standing, removed from bag. 70 c.c. injected intraperitoneally.

10.30 a.m. B.P. 90. P. 150.

100 c.c. removed from rubber bag, fluid now darker in colour, slight odour.

3.0 p.m. B.P. 90. P. 180.

60 c.c. dark coloured fluid removed. Odour more pronounced. 50 c.c. re-injected.

3.30 p.m. B.P. 50. P. 240.

4.15 p.m. Dead. Autopsy showed loop of gut was not perforated.

In this experiment it was possible to remove at intervals the fluid exuding from the loop which collected in the rubber bag. The major portion of the fluid removed was promptly re-injected intraperitoneally and the effect on the blood-pressure and pulse-rate observed. It is noticeable that the earlier injections had very little effect—in fact. the animal's condition showed little alteration in the period between eight and twenty-three hours after the onset of strangulation. But the effect of the injection of the fluid which exuded from the gut between the twentieth and twenty-third hours of

strangulation was prompt and marked, the blood-pressure falling within half an hour and death ensuing one and one quarter hours after the injection. These results were confirmed over a series of cases. It is important to note that the loops were found intact at autopsy; in fact, if the loop strangulated is more than 15 cm. in length, death usually takes place before perforation of the gut occurs. It can be concluded, therefore, that there are present in the fluid exuding from a strangulated loop substances which on absorption from the peritoneum cause death. These substances are not present in the exudate in the early stages, but begin to appear probably about sixteen to twenty hours after the onset and are soon present in sufficient amount to cause death.

3. The Origin of the Toxic Substances Present in the Exudate.—The mode of production of the toxins of acute obstruction has been adequately dealt with by other authors. After a prolonged controversy it is now generally believed that the toxins are the direct result of bacterial action. Dragstedt and his co-workers¹⁷ succeeded in sterilizing short loops of intestine by allowing them to drain freely into the peritoneal cavity, and found that such sterile loops could be strangulated without causing the death of the animal. Murphy and Brooks¹⁸ did the same experiments with similar results and took this to be the final proof that bacteria elaborated the toxin. This view is now held by the majority of investigators. But the object of this section is to discuss the means by which the toxic substances pass from the strangulated loop into the fluid exuding from the loop. It is well known that the lumen contents of a strangulated loop become increasingly toxic after about sixteen hours from the onset of strangulation. It would appear that the lumen contents are therefore the obvious origin of the toxic substances which are found in the exudate and in order to leave the loop in the exudate the toxins must permeate the wall of the intestine. The early exudate from the loop is an odourless, strawberry-coloured fluid which coagulates on standing and is practically indistinguishable from blood-plasma. After about sixteen hours the fluid is found to become darker in colour, later almost black, to be no longer spontaneously coagulable, and to have acquired a faint but definite trace of the odour characteristic of the contents of a strangulated loop. This suggested that after about twenty hours the wall of the intestine was becoming permeable to the loop contents. The permeability of the intestinal wall was investigated by the following experiments:—

Experiment VII.—An enterostomy was performed in such a fashion that whilst one end of the catheter came out on to the abdominal wall the other end passed into the segment of gut to be strangulated. Strangulation was produced by ligating the veins in the mesentery and by including the anastomosing channels at the mesenteric attachment of the bowel within the ligatures which occluded the intestine. The strangulated loop was then enclosed in a rubber bag into the apex of which a rubber tube was fastened and brought out to the abdominal wall. By these means it was possible to obtain samples of the contents of the bowel lumen and of the fluid exuding from the gut simultaneously. At the same time it was possible to measure the pressure within the strangulated loop. In order to have a means of comparing the respective compositions of the fluid exuding from the gut and of the lumen contents the samples were estimated for chloride, sugar, and non-protein nitrogen content. The following tables indicate the results:—

ACUTE STRANGULATION OF INTESTINE 591

		N.P.N.	PLASMA Sugar	Cl	EXUDATE		Cl	GUT CONTENTS ^a		Cl
		N.P.N.	Sugar		N.P.N.	Sugar		N.P.N.	Sugar	
Operation 1.										
5 hrs. later	..	39	108	478	31	86	632	31	30	548
12 hrs. later	..	—	—	—	29	43	640	39	10	528
24 hrs. later	..	43	96	432	56	10	610	82	8	454
Operation 2.										
4 hrs. later	..	23	139	435	25	80	596	32	30	495
11 hrs. later	..	—	—	—	21	26	566	36	19	484
23 hrs. later	..	27	—	—	34	16	508	62	7	474
Operation 3.										
At operation	..	26	178	458	—	—	—	—	—	—
4 hrs. later	..	30	121	408	23	101	622	36	42	532
20 hrs. later	..	36	72	384	34	14	596	49	8	496
Operation 4.										
At operation	..	23	240	514	—	—	—	—	—	—
8 hrs. later	..	17	—	—	26	58	574	69	10	524
20 hrs. later	..	24	234	462	124	12	572	284	9	460
Operation 5.										
At operation	..	22	181	508	—	—	—	—	—	—
8 hrs. later	..	27	197	460	23	87	632	44	13	504
20 hrs. later	..	33	94	450	32	15	614	92	10	502
22 hrs. later	..	—	—	—	38	15	586	106	8	502
Operation 6.										
8 hrs. later	..	—	—	—	24	—	—	58	—	—
20 hrs. later	..	—	—	—	47	—	—	—	—	—
22 hrs. later	..	—	—	—	48	—	—	132	—	—
24 hrs. later	..	—	—	—	55	—	—	—	—	—
26 hrs. later	..	—	—	—	60	—	—	118	—	—

A superficial glance at these results might suggest that the composition of the exudate from the gut follows that of the loop contents. The chlorides remain steady or show a slight fall. The sugar estimation falls in both cases, earlier in the case of the loop contents, probably as a result of bacterial action. More interest centres around the non-protein nitrogen. Whilst a steady rise takes place in the non-protein nitrogen of the loop contents, little change is found in the fluid outside the loop until after the expiration of about twenty hours, at about which time a definite rise in the non-protein nitrogen is noticeable. But even after the expiration of twenty hours the composition of the fluid exuding from the loop is not the same as that in the lumen of the intestine as shown by Operations 5 and 6 in the above group. At the same time an investigation into the pressure existing within the lumen of strangulated loops was carried out. The pressure was very quickly found to rise to between 45 and 75 cm. water. This pressure was maintained for about fifteen to twenty hours and was then found to fall slowly. If the blood-pressure readings are recalled with the sudden and fatal fall occurring about twenty to twenty-four hours after the onset, it would appear that, at this stage, when the lumen pressure is also falling, the wall of the intestine becomes permeable to the toxic substances present in the loop content. This view was held by the writer until the following experiments convinced him that the explanation was not necessarily such a simple one.

592 THE BRITISH JOURNAL OF SURGERY

Experiment VIIIA (May 4, 1932).—Dog. Weight 12 kilos.

3.0 p.m. Operation. $\frac{1}{4}$ gr. morphine and ether anaesthesia. A loop of jejunum 20 cm. long obstructed and its veins ligated. Catheter sutured into loops.

	B.P.	PULSE	LUMEN PRESSURE cm. H ₂ O	LUMEN CONTENTS Fluid	Gas
11.0 p.m. ..	—	—	59	55 c.c.	2 c.c.
11.0 a.m. ..	110	160	47	50 c.c.	18 c.c.
3.0 p.m. ..	112	180	26	47 c.c.	18 c.c.
6.30 p.m. ..	115	220	27	47 c.c.	18 c.c.
10.0 p.m. ..	115	220	27	47 c.c.	18 c.c.
Midnight. Dead. Loop intact.					

Experiment VIIIB (April 11, 1932).—Bitch. Weight 10 kilos.

3.30 p.m. Operation. 25 cm. loop of jejunum obstructed and veins ligated. Catheter sutured into loop.

	B.P.	PULSE	LUMEN PRESSURE cm. H ₂ O	LUMEN CONTENTS Fluid	Gas
3.30 p.m. ..	135	90	—	—	—
11.0 p.m. ..	110	160	36	45 c.c.	—
11.0 a.m. ..	105	220	45	75 c.c.	25 c.c.
2.0 p.m. ..	105	220	40	70 c.c.	30 c.c.
5.0 p.m. ..	100	240	30	65 c.c.	35 c.c.
7.0 p.m. Dead. Loop intact.					

Experiment VIIIC (May 4, 1932).—Bitch. Weight 10 kilos.

3.45 p.m. Operation. $\frac{1}{4}$ gr. morphine and ether anaesthesia. Veins to a loop of small intestine 25 cm. long ligated. Loop obstructed and catheter sutured into loop in manner previously described. Blood-pressure readings obtained from femoral artery.

	B.P.	PULSE	LUMEN PRESSURE cm. H ₂ O	LUMEN CONTENTS Fluid	Gas
11.0 p.m. ..	120	160	29	20 c.c.	8 c.c.
12.0 noon ..	112	200	43	15 c.c.	20 c.c.
3.15 p.m. ..	110	240	29	15 c.c.	20 c.c.
7.0 p.m. ..	85	240	30	8 c.c.	22 c.c.
10.0 p.m. Dead. Autopsy showed the loop only slightly distended but not perforated.					

Experiment VIIID (April 11, 1932).—Dog. Weight 10 kilos.

2.30 p.m. Operation. $\frac{1}{4}$ gr. morphine and ether anaesthesia. Loop of jejunum 25 cm. long about 30 cm. below Treitz ligament obstructed and veins ligated. Catheter sutured into loop which was enclosed in a rubber bag draining by a tube on to the surface.

	B.P.	PULSE	LUMEN PRESSURE cm. H ₂ O	LUMEN CONTENTS Fluid	Gas
4.30 p.m. ..	—	—	70	25 c.c.	—
10.0 p.m. ..	140	60	70	25 c.c.	10 c.c.
11.30 a.m. ..	130	160	70	15 c.c.	40 c.c.
12.0 noon ..	125	160	70	10 c.c.	40 c.c.
2.30 p.m. ..	110	170	65	12 c.c.	43 c.c.
4.15 p.m. ..	95	190	47	12 c.c.	43 c.c.
4.35 p.m. Dead. Autopsy again showed an intact loop.					

The first point to be considered in the above experiments is the pressure registered in the lumen. Fluid is poured into the lumen of the loop shortly after strangulation is produced, and the volume apparently reaches its

maximum about eight to twelve hours after the onset, and some time later, about twenty hours after the onset, there appears to be a tendency for the fluid to leave the lumen. It would seem that the early pressures are independent of the volume of the loop contents and probably are indicative of the tonic contractions which are known to take place after strangulation has been produced and to last for several hours.¹³ But as the pressure falls there is no corresponding reduction in the volume of the loop contents—in fact, there appears to be an increase in the volume of the loop contents owing to the production of gas taking place at the very time when the loop pressure is falling. In none of the above experiments could it be shown that any considerable volume of fluid passed out of the lumen before death took place.

Various experiments were carried out in which substances easily recognizable by colour reactions such as phenolsulphonaphthalein and potassium ferricyanide were introduced into the lumen of the loop at the time of strangulation. The presence of these substances was never demonstrable in the exudate from the loop, but at the time this failure was attributed to technical difficulties associated with the high protein content of the exudate. The experiments have more significance when considered with the convincing results published by Scott and Wangenstein on the absorption of strychnine sulphate from strangulated loops of intestine.¹⁹ They were unable to show that strychnine passed out of strangulated loops in the absence of perforation of the intestinal wall. But it must be noted that in their experiments such long loops of gut were used that death followed in about eight to ten hours.

In an attack on the problem from another angle the following experiments were performed in which the contents of the lumen were removed at intervals and replaced by a corresponding volume of air or water.

Experiment IX A (May 16, 1932).—Bitch. Weight 9 kilos.

- 10.15 p.m. Operation. $\frac{1}{4}$ gr. morphine and ether anaesthesia. Loop of jejunum 25 cm. long obstructed and veins ligated. Catheter sutured into loop.
- Noon. Following day. Contents removed and, after the loop had been washed out several times with water, they were replaced by 60 c.c. air.
- 4.30 p.m. B.P. 105. Pulse 200. Lumen pressure 50 cm. Loop found to contain 55 c.c. gas and 10 c.c. fluid. Replaced by equal volume air after washing of loop.
- 11.0 p.m. B.P. 100. Pulse 240. Lumen pressure 53 cm. Loop found to contain 67 c.c. gas and 3 c.c. of fluid. Loop washed and 70 c.c. air injected.
- 6.30 a.m. B.P. 95. Pulse 240. Lumen pressure 50 cm. Dead. Autopsy showed loop intact.

Experiment IX B (May 24, 1932).—Dog. Weight 9 kilos.

- 10.30 p.m. Operation. About 25 cm. ileum obstructed and veins ligated. Catheter sutured into loop.
- 11.0 p.m. Loop found to contain 16 c.c. fluid and 44 c.c. gas. Replaced by same volume air.
- 3.0 p.m. Loop found to contain 62 c.c. gas and 3 c.c. fluid. Replaced by water.
- 11.0 p.m. Dog very sick.
- 8.0 a.m. Found dead. Cold and evidently been dead for several hours. Loop not perforated.

- Experiment IX C* (June 7, 1932).—Dog. Weight 9 kilos.
 10.30 a.m. Operation. Loop of ileum 20 cm. long obstructed and veins ligated.
 Catheter sutured into loop.
 10.30 p.m. 28 c.c. of fluid removed from loop and replaced by air.
 10.30 a.m. Contents replaced by water.
 10.30 p.m. Dog very sick.
 8.0 a.m. Found dead. Cold and evidently dead several hours.

Six of these experiments were carried out, and in none of them could it be shown that life had been prolonged as a result of the removal of the loop contents. Furthermore, a comparison of the amount of fluid exuding from a strangulated loop and the volume of fluid in the lumen brings out the following figures:—

Experiment X A.—

HOURS STRANGULATED	TOTAL VOLUME OF EXUDATE c.c.	VOLUME OF LOOP CONTENTS	
		Gas	Fluid
2	10	—	25
8	36	10	25
20	160	40	15
22	190	40	10
24	208	43	12
26	223	43	12

Experiment X B.—

8	35	—	45
20	100	25	75
22	135	30	70
24	160	—	—
26	175	35	65

The large volume of fluid exuding from a strangulated loop during twenty-four hours is well shown in the above figures. During the period between the eighth and twenty-sixth hour of strangulation 188 c.c. were exuding from the loop whilst 13 c.c. were passing from the lumen in the first experiment. Corresponding figures for the second experiment are 140 and 10. In the writer's experience the lumen content of a loop strangulated for twenty four hours has not been sufficiently highly toxic to cause death when reaching the peritoneum in such small amounts over such a period. It would appear, therefore, that the toxic substances shown to be present in the exudate from the loop are not derived mainly from the loop contents. They must, therefore, be formed in the wall of the intestine.

4. **Conclusion.**—When a loop of intestine of moderate length (20 to 40 cm. in the average dog) is strangulated insufficient blood is withdrawn from the circulation to cause death. The animal lives long enough for the intestinal bacteria to invade the tissues of the intestinal wall and produce toxic substances which pass out of the strangulated loop into the peritoneal cavity and when absorbed cause death. These toxins are present in the exudate from the strangulated loop in increasing amounts after about sixteen hours from the onset of strangulation. The toxins in the exudate are produced in the wall of the intestine and do not result from the permeation of the intestinal wall by the toxic substances present in the contents of the intestinal lumen.

III. THE STRANGULATION OF SHORT LOOPS.

The strangulation of short loops of intestine provides still another problem. The withdrawal of blood from the circulation into the loop is small and the amount of toxin present in the exudate from the loop is insufficient to cause death rapidly. As a result the disintegration of the intestinal wall progresses until perforation takes place and the contents of the lumen escape into the peritoneal cavity. The absorption of the additional toxin from the lumen contents may be sufficient to cause death soon after the loop perforates. If not, the animal develops a widespread peritonitis from which death quickly ensues. There still remains for investigation the group of cases in which only a knuckle of gut is strangulated. With these very small loops interesting results are obtained :—

Experiment 1 (April 25, 1932).—Bitch. Weight 10 kilos.

2.30 p.m. Operation. $\frac{1}{4}$ gr. morphine and ether anaesthesia. 5 cm. loop of jejunum obstructed and vein ligated.

April 26.—

12.15 p.m. B.P. 115. Pulse 140. Dog quiet but able to walk. Given $\frac{1}{4}$ gr. morphine.

2.30 p.m. B.P. 105. Pulse 70. Able to run about.

4.15 p.m. B.P. 105. Pulse 80.

10.45 p.m. B.P. 110. Pulse 180.

April 27.—

Noon. B.P. 90. Pulse 220. Mass palpable in upper left abdominal quadrant. Abdomen apparently tender on examination.

2.45 p.m. Dead. Autopsy. No evidence of general peritonitis. Well localized cavity formed by coils of small intestine and omentum. Strangulated loop occupied centre of abscess cavity. Loop perforated and surrounded by grumous fluid. No free fluid in peritoneum. Distension of proximal loop.

Experiment 2 (April 25, 1932).—Bitch. Weight 10 kilos.

1.30 p.m. Operation. $2\frac{1}{2}$ cm. loop of jejunum obstructed and veins ligated.

April 26.—

Noon. B.P. 105. Pulse 160. Given $\frac{1}{4}$ gr. morphine. Dog able to walk but quiet.

2.0 p.m. B.P. 105. Pulse 120.

4.0 p.m. B.P. 105. Pulse 200.

11.0 p.m. B.P. 100. Pulse 200.

April 27.—

Noon. B.P. 110. Pulse 180. Dog quite strong. Washed himself during morning. No mass palpable on examination.

3.0 p.m. Laparotomy. Fair quantity of brown odourless fluid in peritoneum. Small omental mass found under left costal margin. Early and slight injection of mesentery and intestine. Jejunal loop found at centre of abscess cavity in omentum. Evident that dog had early peritonitis. Dog destroyed.

The first of these dogs lived forty-eight hours—at least twelve hours longer than any dog in the writer's series of strangulation of medium-sized loops. This animal had no widespread peritonitis, but died from the absorption of toxins from the abscess cavity, in the centre of which was found the perforated strangulated loop. The abscess cavity bore a striking resemblance

to that provided by an acute gangrenous appendicitis of the obstructive type in the human being. The second dog was in fairly good condition when it was destroyed over forty-eight hours after strangulation was produced. This dog had a very small loop strangulated, and the blood-pressure and pulse rate on the second day show how well the animal was able to cope with the small amounts of toxic substances produced by such a small loop. This dog had definite evidence of a spreading peritonitis and would have died probably within the next twenty-four hours.

All the experiments which have been considered up to date have dealt with internal strangulation—that is to say, where the loop is free within the peritoneal cavity. But so many strangulations in actual practice originate in a hernia, and the constriction at the neck of the hernial sac which produces strangulation also shuts off the contents of the sac from the general peritoneal cavity. It is well known that internal strangulation is more rapidly fatal than a corresponding external strangulation.²⁰ This is undoubtedly due to the fact that absorption from a hernial sac is much slower than from the peritoneal cavity. The capability of a dog to withstand the absorption of small amounts of the toxin is well shown in the last experiment. Forty-seven hours after strangulation this dog had a blood-pressure and pulse-rate very little changed from those taken twenty-four hours after strangulation. Yet this loop was intra-abdominal. It is quite certain that, had this loop been enclosed in a femoral hernial sac, the dog's condition would have been even better and there would have been no general peritonitis. Chenut¹⁴ showed that life was prolonged in strangulation when the loop was placed extraperitoneally. It is along these lines that an explanation is to be found for the cases of strangulated femoral hernia of five or more days' duration which occasionally present themselves at general hospitals. The amount of toxin produced is not large and absorption is slowed to such an extent that the body is able to withstand the effects of its absorption. The strangulated loop forms a localized abscess the effects of which the body might conceivably survive were it not for the association of obstruction of the proximal portion of the small intestine. If left untreated, this in itself is capable of causing death. It is this obstruction which in late cases of small strangulated femoral herniae is probably the main lethal factor.

STRANGULATION IN WHICH TOTAL ANÆMIA EXISTS.

This condition was investigated in 1924 by Foster and Hausler¹⁰ and later by Wangenstein and Scott.²¹ The condition occurs in practice very rarely, if at all. There is no blood lost into the loop and the acute collapse symptoms of the venous obstruction series are not shown. The loop is found at autopsy sometimes perforated, at other times intact but distended with gas. Well-marked evidence of a general peritonitis is always present at autopsy. The wall of the devitalized intestine is rapidly invaded by the intestinal flora. The loop usually becomes distended but from a different cause—the activity of gas-producing organisms. This distension has the effect of expressing fluid from the disintegrating tissues into the abdominal cavity. The fluid carries with it not only the products of the protein

metabolism taking place in the tissues but also the organisms responsible for the proteolysis. As a result the animal succumbs to a combination of a toxæmia, resulting from absorption of substances from the loop, and a generalized peritonitis.

THE NATURE OF THE TOXIN PRODUCED IN STRANGULATED LOOPS OF INTESTINE.

It is now universally accepted that the presence of bacteria in the lumen of the intestine is necessary for the production of the lethal substances which produce the toxæmia of acute intestinal strangulation. The bacterial origin of the toxin has been proved by the investigations of Dragstedt,¹⁷ Murphy,¹⁸ and their associates. The exact nature of the toxin is still doubtful. During recent years the interest of research workers has centred around the possibility that heat-labile toxins are the cause of the toxæmia of acute strangulation. This followed a paper published by Williams²² in 1926, in which he suggested that the toxin produced by the *B. welchii* was, at least in part, the lethal agent in acute obstruction. McIver, White, and Lawson¹³ tested this theory in relation to acute strangulation. They found from cultural studies of the bacterial flora of strangulated loops of bowel in cats that the number of *B. welchii* was always so increased that there was no doubt that this was the predominant organism. Further experiments showed that the organisms obtained were generally avirulent strains, and the administration of large doses of the *B. welchii* antitoxin failed to prolong the lives of animals with experimental acute strangulation. Other workers experimenting with a different type of acute obstruction were also unable to show that any benefit resulted from the use of the *B. welchii* antitoxin.²³⁻²⁵ In consequence, it is no longer believed that the toxin of the *B. welchii* plays any important part in the toxæmia of acute intestinal obstruction. Williams²² also made the very pertinent suggestion that the methods employed by many of the earlier workers,²⁶⁻²⁸ which usually included heating of their preparations for varying periods, might result in the liberation of a toxic substance. He tested out his theory by heating for one hour the material obtained from patients and injected the Berkefeld filtrate into mice. His results suggested that a toxic substance was liberated from the intestinal contents by heating. My own experiments do not support this contention.

In these experiments the material was obtained from dogs in which an experimental strangulation had been produced. This choice was made because: (1) It had been previously shown that the fluid exuding from loops of intestine which had been strangulated for twenty hours or more contained toxic substances which caused death on absorption from the peritoneal cavity; and (2) A strangulated loop provides ideal cultural conditions for anaerobic organisms. The fluid used for injection was obtained in one of two ways: (1) From loops of intestine in which venous strangulation had been produced; or (2) By extracting with cold saline or water the tissues of intestinal loops to which both artery and veins had been tied. The loops of the venous strangulation series were all enclosed in rubber bags and left for a specified time in the abdominal cavity. In the arterial series the loops were either

incubated in rubber bags in the abdominal cavity or were incubated anaerobically *in vitro* at 37° C. Where filtration was carried out it was done in the cold, using a Berkefeld filter (gauge N). All injections were made intraperitoneally and small dogs were used for the purpose. The results are divided into two groups: (1) Those in which the fluid was obtained from loops in which a venous strangulation had been produced; and (2) Those in which an extract from loops with an arterial strangulation was used.

Results in Group I.—

	HOURS INCUBATION	FILTERED	BOILED	RESULT	
1	24	—	—	Dead	10 hrs.
2	24	—	Yes	Dead	4½ "
3	24	—	—	Dead	7 "
4	24	—	—	Dead	6 "
5	24	Yes	—	Well	—
6	24	Yes	—	Well	—
7	24	Yes	Yes	Well	—
8	24	Yes	Yes	Well	—

Results in Group II.—

	INCUBATED	INCUBATED	FILTERED	BOILED	RESULT	
1	24	In vivo	—	—	Dead	6 hrs.
2	24	In vivo	—	—	Dead	10 "
3	36	In vivo	Yes	—	Well	—
4	36	In vivo	Yes	Yes	Well	—
5	24	In vitro	Yes	—	Well	—
6	24	In vitro	Yes	Yes	Well	—
7	24	In vitro	Yes	—	Well	—
8	24	In vitro	Yes	Yes	Well	—
9	48	In vitro	Yes	—	Well	—
10	48	In vitro	Yes	Yes	Well	—
11	48	In vitro	Yes	—	Well	—
12	48	In vitro	Yes	Yes	Well	—
13	24	In vitro	—	Yes	Well	—
14	24	In vitro	—	—	Dead	3 hrs.
15	24	In vitro	Yes	—	Dead	5 "

These results show quite definitely that Berkefeld filtration removes the toxic substances from the fluid. In one experiment only, No. 15 of Group II, was there a fatal result following the injection of a Berkefeld filtrate. In seven of the experiments the filtrate was boiled before the injection was made without any evidence of increased toxicity being demonstrated. All the animals very shortly after the injection was made showed well-marked evidence of histamine poisoning—namely, shivering, tremors of the hind limbs, salivation, vomiting, urination, and defæcation. These effects passed off within about half an hour and did not recur even in the animals which died.

These results are similar to those published by McIver and his associates.¹³ They obtained their material from strangulated loops and injected various fractions intravenously into cats. They concluded that: (1) The crude loop contents were highly toxic; (2) The sediment obtained by centrifugation was highly toxic, but the supernatant fluid varied considerably in toxicity even though both the crude loop contents and the sediment from the same

animal were very toxic; (3) The filtrate resulting from Berkefeld filtration had no depressor effect on the blood-pressure. They also prepared a 'dialysate' obtained by removing the strangulated loop from the animal, suspending it in physiological salt solution and placing it in the incubator for two hours. The 'dialysate' was found to have a decided depressor effect and its toxicity was not destroyed by boiling for five minutes. These results appear to support the contention of the earlier workers who reported that the toxin was not destroyed by heat^{6, 18, 29} and did not pass through a Berkefeld filter.²⁰ The toxin, though it is precipitated by five volumes of alcohol,²⁸ possesses no antigenic properties.³¹ There is a striking similarity between the pharmacological actions of the toxic substance and those of the proteoses,^{32, 33} and most workers³⁴ are agreed that the toxin is probably a proteose.

COMMENTARY.

From these experiments it is evident that the cause of death in venous strangulation depends directly on the length of bowel affected. With the onset of strangulation the loop of intestine becomes engorged with blood, rapidly at first but later decreasing as thrombosis spreads in the capillaries and small arterial twigs. The tissue spaces become distended with plasma and later with blood resulting from ruptured capillaries. When the loop of intestine is of sufficient length death takes place as a result of the rapid withdrawal of blood from the circulation. With loops of shorter length insufficient blood is lost to cause death, and thus other factors enter into the pathology. Quite soon after strangulation is produced the mucosa is invaded by the rapidly proliferating intestinal bacteria. The organisms spread to the deeper layers and find in the plasma filling the tissue spaces an ideal culture medium. The proteolytic anaerobes are the organisms which increase most rapidly in numbers. Meanwhile the loop of intestine is so distended with blood that large amounts of plasma pass through the peritoneal covering giving rise to the exudate which plays such an important part in the pathology of this condition. The exudate continues long after thrombosis is complete in the vessels of the loop.

The pressure within the lumen of the strangulated loop which was at first mainly due to a tonic contraction of the intestinal musculature is maintained, as the muscle dies, by the gas resulting from the action of the intestinal flora. It is probable that the distension of the loop which results from this gas production provides the mechanism by which the exudate from the loop is continued in the later stages. The exudate, which is at first indistinguishable from blood plasma, later becomes darker in colour and acquires a foetid odour. An alteration in the toxicity of the fluid coincides with this change in appearance. The early exudate is non-toxic, but the exudate leaving the loop about twenty hours after the onset of strangulation is very toxic on absorption. At first it was thought that the exudate acquired its toxicity as the result of the permeation of the intestinal wall by the toxic substances present in the loop contents. Experimental evidence has been produced to show that it is doubtful if such permeation takes place, and, if it does, it is definitely insufficient in quantity to influence the result. The

toxic quality of the exudate in the later stages is the direct result of the action of the proteolytic organisms which have invaded the intestinal wall and tissue spaces in large numbers from the lumen. Chief amongst these organisms is the *B. welchii*, but the toxic qualities of exudate appear to be due to substances akin to the proteoses and not to any bacterial exotoxin. It is the absorption of the toxic exudate from the loop via the peritoneal cavity which is the cause of death in strangulation of intestinal loops of moderate length. When a small loop is strangulated the course of events proceeds a stage further. The amount of toxin escaping in the exudate is insufficient to cause death, and the disintegration of the intestinal wall continues until perforation takes place. Death may then occur as a result of the absorption of the toxic loop contents which have escaped into the peritoneal cavity, or from the widespread peritonitis which rapidly ensues. When very small loops of intestine are strangulated in extraperitoneal positions, e.g., femoral hernia, evidence has been brought forward which suggests that the dehydration resulting from the accompanying obstruction of the small intestine may be the lethal factor.

CLINICAL CONSIDERATIONS.

The subject matter of this paper has so far dealt solely with experimental strangulation. It is intended to indicate in this section the lines along which the results of the investigations already mentioned may be applied to the treatment of acute strangulation as it occurs in man. In all the experiments described in this paper strangulation has been produced by ligation of the mesenteric veins and, consequently, has been acute and complete. These conditions are not always—perhaps not even frequently—reproduced in clinical practice. In a typical case of strangulated hernia it is not usual for the venous drainage of the incarcerated loop of intestine to be suddenly and completely obstructed. At first there is a mild degree of venous obstruction which leads to some congestion and œdema of the intestine and mesentery which further obstructs the veins. In this way a vicious circle is set up which results in the venous obstruction becoming complete and the condition of strangulation developed. The case in which strangulation is present from the onset is usually characterized by such acute symptoms as to be unmistakable. It is obvious, therefore, that the various changes taking place in a strangulated loop will occur later in practice than is the case in experimental strangulation. This explains the apparent rapidity of the sequence of events in experimental strangulation.

An acute strangulation of the intestine in man which is comparable to the experimental long-loop strangulations is very uncommon. But there occur with reasonable frequency three allied conditions which, though generally not considered as such, are in effect strangulations. These are acute intussusception, mesenteric thrombosis, and certain cases of acute volvulus. In these conditions an interference with the blood-supply of a segment of intestine is an integral part of their pathology. In acute intussusception the venous drainage of the intussusception is obstructed before the condition has progressed very far, the intestine is acutely congested, and blood escapes into the lumen of the intussusciens, later producing the typical bloody

stools. In mesenteric thrombosis the venous obstruction takes some time to become complete, but again blood is lost into the loop. In acute volvulus the venous drainage may be completely obstructed at the time of the production of the volvulus, or the increasing distension of the loop may completely obstruct an already impaired venous drainage. But these conditions present two common features: (1) Long lengths of bowel may be affected; (2) A complete venous obstruction exists in the length of bowel affected. The blood loss, which is so marked a feature of the experimental long-loop strangulation, is equally evident in these clinical conditions. The shock which was previously attributed to twisting of the mesentery, pain, etc., has a more reasonable explanation. It is due to the withdrawal of blood from the circulation by the affected segment of intestine, and a blood transfusion is therefore an essential part of any treatment adopted.

The moderate-sized loops described in this paper have possibly more frequent clinical counterparts. They are still too long to occur frequently in strangulated external hernia, but do occur as internal strangulations. The cause of death in such cases has been shown to be the absorption of toxic products, formed in the wall of the intestine, from the peritoneal cavity. The danger of excessive manipulation of such loops lying within the abdomen is at once evident and the practice of exteriorizing such a loop immediately on discovery cannot be too strongly recommended.

The short loops occur frequently in clinical practice and fortunately present themselves as strangulated external herniæ and consequently receive prompt treatment. The fatal cases are usually those where there has been considerable delay in seeking treatment and the strangulated loop has become gangrenous and perforated, resulting usually in a widespread peritonitis.

There still remains a form of acute strangulation, which, though already mentioned, merits special consideration. This is the very small strangulated hernia, usually femoral, in which only a very small segment of bowel is affected or when the whole circumference of the gut is not involved, e.g., a Richter's hernia. In such a condition there is neither sufficient blood lost into the loop nor toxins absorbed from the loop to affect the patient's condition to any extent. Furthermore, the neck of the sac and the strangulated gut usually become sufficiently adherent to limit for the time being the infection which results from the perforation of the loop and which would otherwise spread back into the general abdominal cavity and lead to a generalized peritonitis. Such a patient may go on for several days until persistent vomiting and colicky abdominal pains force him to seek treatment. His general condition may then be very poor and surgical treatment a hazardous proceeding. But it should be borne in mind that his low general condition is the result of the obstruction of the intestine which naturally accompanies the strangulation. In the more massive strangulations the effect of superimposed obstruction is negligible, but in the type of case mentioned above it becomes of supreme importance. The dehydration and chloride loss from repeated vomiting may assume such proportions as to become in themselves lethal factors. It is necessary in all such cases to combat the state of dehydration by the administration of adequate amounts of sodium chloride solution before surgical interference is considered.

The actual surgery of acute strangulation depends on the state of the intestine at operation. Any certain test of the viability or otherwise of strangulated intestine would be invaluable. Perhaps the most reliable of the various tests which have been used experimentally is the response of the gut to a strong faradic current. Intestine which fails to respond is definitely non-viable, but the converse does not always hold, and the test, in any case, is not likely to be generally useful. Any loop from which the peritoneal exudate has a fœtid odour should be regarded as non-viable. But experimental work has no reliable test of viability of strangulated intestine to offer. The time-honoured tests must be applied and one of two decisions reached. Gut must be regarded as definitely viable or non-viable. All doubtful cases should be regarded as non-viable, and under no circumstances should a doubtful loop of gut be returned to the abdomen. Such a loop should be brought out of the abdomen and a Paul's operation performed—in ill patients the loop may be excised the day following the operation. I believe it is safer to carry out such a procedure rather than attempt an immediate anastomosis, but this point is of secondary importance to the principle that all strangulated loops of doubtful viability should be resected.

CONCLUSIONS.

1. The duration of survival following venous strangulation of the intestine varies directly with the length of bowel affected.

a. When long loops are strangulated there is no evidence of any toxæmic process and death is due solely to the withholding of a large volume of blood from the circulation by the loop.

b. When moderate-sized loops of intestine are strangulated insufficient blood is withheld from the circulation to bring about a fatal issue, and death now results from the absorption, via the peritoneal cavity, of the fluid exuding from the loop, which becomes highly toxic about twenty hours after the onset of strangulation.

c. When short loops are strangulated there is neither sufficient blood withheld from the circulation nor toxins absorbed to cause death. Perforation of the loop always precedes death, which follows from the resulting widespread peritonitis aided by the absorption of the toxic loop contents. Only rarely will the latter be the sole agency to bring about a fatal result.

2. The toxic qualities of the peritoneal exudate from a strangulated loop result from the action of proteolytic organisms on the tissues of the intestinal wall. The exudate is not derived from the contents of the lumen of the loop.

3. There is no support for the view that bacterial exotoxins (e.g., *B. welchii*) are responsible for the toxæmia of acute strangulation. Evidence is brought forward which suggests that the toxin is of large molecular form and probably akin to the proteoses.

4. It is suggested that, when very small loops are strangulated in extra-peritoneal positions, e.g., femoral hernia, the cause of death is the dehydration resulting from the obstruction of the small intestine which accompanies the strangulation.

5. When the arteries as well as the veins are obstructed in strangulation death follows from a combination of toxæmia and peritonitis resulting from the action on and the penetration of the intestinal wall by bacteria from the lumen.

The experimental work described was carried out at the Johns Hopkins Hospital, Baltimore, U.S.A., under the direction of Professor Dean Lewis and Dr. Lee during the tenure of the Dickinson Travelling Scholarship in Medicine.

REFERENCES.

- ¹ BROOKS, B., SCHUMACHER, H. W., and WATTENBERG, J. E., *Ann. of Surg.*, 1918, lxxvii, 210.
- ² HARTWELL, J. A., *Jour. of Exper. Med.*, 1913, xviii, 139.
- ³ HOWELL, J., *Brit. Med. Jour.*, 1913, ii, 1645.
- ⁴ MURPHY, F. T., and VINCENT, BETH, *Boston Med. and Surg. Jour.*, 1911, clxv, 684.
- ⁵ REICHEL, P., *Dent. Zeits. f. Chir.*, 1892-3, xxxv, 495.
- ⁶ SUGITO, S., *Mitteil. a. d. Med. Fak. d. k. Univ. Kyushu Fukuoka*, 1924, ix, 229.
- ⁷ WILKIE, D. P. D., *Brit. Med. Jour.*, 1913, ii, 1064.
- ⁸ VON ALBECK, *Arch. f. klin. Chir.*, 1902, lkv, 569.
- ⁹ EISBERG, H. B., *Ann. of Surg.*, 1921, lxxiv, 384.
- ¹⁰ FOSTER, W. C., and HAUSLER, R. W., *Arch. of Internal Med.*, 1924, xxxiv, 697.
- ¹¹ COPPER, G. H., and BROOKS, B., *Ann. of Surg.*, 1923, lxxviii, 755.
- ¹² BLALOCK, A., *Arch. of Surg.*, 1930, xx, 959.
- ¹³ MCIVER, M. A., and WHITE, J. C., *Ann. of Surg.*, 1929, lxxxix, 647.
- ¹⁴ CHENUT, A., *Rev. de Chir.*, 1926, lxiv, 474.
- ¹⁵ SCOTT, H. G., and WANGENSTEEN, O. H., *Proc. Soc. Exper. Biol. and Med.*, 1932, xxix, 748.
- ¹⁶ SCOTT, H. G., and WANGENSTEEN, O. H., *Ibid.*, 559.
- ¹⁷ DRAGSTEDT, L. R., DRAGSTEDT, C. A., MCCLINTOCK, J. T., and CHASE, C. S., *Jour. of Exper. Med.*, 1919, xxn, 109.
- ¹⁸ MURPHY, F. T., and BROOKS, B., *Arch. of Internal Med.*, 1915, xv, 392.
- ¹⁹ SCOTT, H. G., and WANGENSTEEN, O. H., *Proc. Soc. Exper. Biol. and Med.*, 1932, xxix, 287.
- ²⁰ SCHONBAUER, L., *Arch. f. klin. Chir.*, 1924, cxxx, 427.
- ²¹ SCOTT, H. G., and WANGENSTEEN, O. H., *Proc. Soc. Exper. Biol. and Med.*, 1932, xxix, 424.
- ²² WILLIAMS, B. W., *Brit. Jour. Surg.*, 1926, xiv, 295.
- ²³ NORTH, J. P., *Ann. of Surg.*, 1930, xcii, 277.
- ²⁴ OWINGS, J. C., and MCINTOSH, C. A., *Arch. of Surg.*, 1929, xviii, 2237.
- ²⁵ THURSTON, H. F., *Ibid.*, 1931, xxii, 72.
- ²⁶ DRAGSTEDT, C. A., DRAGSTEDT, L. R., and CHASE, C. S., *Amer. Jour. Physiol.*, 1918, xlvi, 366.
- ²⁷ DAVIS, D. M., and MORGAN, H. S., *Bull. Johns Hopkins Hosp.*, 1914, xxv, 39.
- ²⁸ ELLIS, J. W., *Ann. of Surg.*, 1922, lxxv, 429.
- ²⁹ WHIPPLE, G. H., *Jour. Amer. Med. Assoc.*, 1915, lxx, 476.
- ³⁰ WERELIUS, A., *Ibid.*, 1922, lxxix, 535.
- ³¹ DRAGSTEDT, C. A., and MOORHEAD, J. J., *Jour. of Exper. Med.*, 1918, xxvii, 359.
- ³² WHIPPLE, G. H., RODENHAUGH, F. H., and KILGORE, A. R., *Ibid.*, 1916, xxiii, 123.
- ³³ CHITTENDEN, R. H., MENDEL, L. B., and HENDERSON, Y., *Amer. Jour. Physiol.*, 1898, ii, 142.
- ³⁴ WILKIE, D. P. D., *Lancet*, 1922, i, 1135.

NEUROSURGERY IN THE TREATMENT OF DISEASES OF THE PERIPHERAL BLOOD-VESSELS.

BY D. J. MACMYN,

FELLOW IN NEUROSURGERY, THE MAYO FOUNDATION; FELLOW OF THE ROCKEFELLER FOUNDATION.

MANY contributions have been made during the last decade to the study and treatment of peripheral vascular disease. Since the pioneer work of A. W. Adson and his associates at the Mayo Clinic a new field in surgery has been opened. One of the earliest operations on the sympathetic nervous system was performed by Jaboulay in 1899, when he attempted to relieve painful conditions of the lower extremities by performing peri-arterial sympathectomy. Leriche brought this procedure to the notice of surgeons in 1913, when he advocated the operation for numerous peripheral vascular, trophic, and painful lesions. Following the work of Hunter, who postulated the sympathetic innervation of striated muscles in animals, Royle advocated ramisection for the relief of spasticity in cases of spastic paralysis. In attempting to duplicate the successful results which Royle reported in his series, Adson performed his first abdominal transperitoneal lumbar sympathetic ganglionectomy on May 20, 1924. He believed that some reduction of the spasticity was obtained, but attributed this to the increased blood-supply to the extremities following the complete interruption of vasomotor constrictor fibres. He based his operation on the belief that a more thorough denervation could be obtained in this way than by section of the rami communicantes alone. Experience has shown that this belief has been amply confirmed. However, as Adson has stated, "If the vasomotor fibres are divided by ramisection or by ganglionectomy and trunk resection it is immaterial which method is used so long as they are completely resected". The operation substantiates also the observations of Kramer and Todd, Potts, and Woollard that the innervation of the arteries and veins does not run centrifugally along the vessels, but that the vessels receive their innervation at various levels corresponding to the musculo-cutaneous segments.

It was following this operation that Adson made the historical observation that the temperature of the skin of the lower extremities remained permanently increased. This evidence of the change in temperature, which was due to an increased blood-flow following vasodilatation, led him to suggest that the operation might be applied to the relief of certain vasospastic diseases. Confirmation was first obtained by the pre-operative and post-operative calorimetric studies made by G. E. Brown on patients with spastic paralysis on whom Adson has performed sympathetic lumbar ganglionectomy and trunk resection. This report was encouraging in that it showed there was an average increase of 400 per cent in the elimination of heat following operation, and that the increase of heat production was permanent. Consequently

the operation was carried out for the first time by Adson for a case of Raynaud's disease of the lower extremities on March 19, 1925. The result was highly successful. Subsequent study showed that permanent vasodilatation was obtained and that the patient's symptoms were completely relieved.

Another contribution to the surgery of the sympathetic nervous system which was equally important was made by Adson in 1928, when he performed a bilateral cervico-thoracic ganglionectomy and trunk resection by the posterior intrathoracic approach. This approach was a modification of that first suggested by A. K. Henry in 1924 for the resection of the left cervico-thoracic ganglion in angina pectoris. The operation has entirely replaced the anterior or Jonnesco approach at the Mayo Clinic and elsewhere.

Following the successful results obtained in patients suffering from Raynaud's disease, the operation was extended to other diseases in which vasospastic phenomena were the outstanding features. It thus included thrombo-angiitis obliterans (Buerger's disease), in which the characteristic finding is occlusion of the main vessels associated with vasospasm of the collateral circulation. More recently it has been applied to certain types of scleroderma and chronic infective arthritis, in which a vasospastic element is present, and for subjective relief in anterior poliomyelitis.

In the wave of enthusiasm which has followed the publication of the successful results of treatment by sympathetic neurectomy, there have been instances in which the results have not been satisfactory. These have been mainly due to the selection of unsuitable cases. As Adson and Brown have stated, "With the advent of surgical procedures capable of producing arterial dilatation in the extremities, it becomes highly important to define clearly the types of vascular diseases that may be benefited by vasodilator measures. Considerable discrimination and caution should be employed in the selection of operable cases". A study of a large series of cases in which operation has been performed during the last eight years has enabled them to formulate their opinions regarding the selectivity of cases, and these have again been more recently expressed by W. J. Mayo and Adson. They will be mentioned briefly below.

At the Mayo Clinic all cases are carefully studied and classified, not only on the condition of their vascular system and the response to vasodilating agents, as determined by cutaneous temperature studies, but on the present status and course of the disease.

The two diseases with which we are mainly concerned from a surgical standpoint are thrombo-angiitis obliterans and Raynaud's disease.

THROMBO-ANGIITIS OBLITERANS.

(Buerger's Disease.)

Thrombo-angiitis obliterans occurs almost exclusively in men between the age of 20 and 50 years. The youngest case reported at the Mayo Clinic was 17, the oldest 73 years. These cases are exceptional. Until lately it was thought that the disease occurred almost exclusively in Jews, but a recent analysis has shown that only 45 per cent of the cases occurred in Jews.

The majority of the patients are cigarette-smokers, and in a recent publication it has been shown by skin tests that they are unusually sensitive to tobacco. Furthermore, it has been shown by Barker that a certain degree of vasoconstriction occurs in normal persons during and after the smoking of several cigarettes. However, 5 per cent of cases of thrombo-angiitis obliterans have never smoked, while there are relatively very few cigarette-smokers who develop the disease. In a series of 240 cases the disease involved the lower extremities in 60 per cent, it affected the upper extremities alone in 2 per cent, and both upper and lower extremities in 38 per cent.

Superficial acute phlebitis is frequently seen in the course of the disease. Buerger and, more recently, Horton were able to produce inflammatory vascular changes in animals experimentally, by implantation of acutely inflamed veins. The investigations on the etiology of the disease are not yet conclusive. Adson is of the opinion that vasospasm may be the initial precipitating factor leading subsequently to thrombosis. In support of this, it has been observed that vasospasm, as evidenced by a phase in which the fingers or toes "go white and cold", is one of the features which is sometimes noted at the onset of the disease; while in another group of typical thrombo-angiitis obliterans of the lower extremities subsequent or coincident changes are seen in the vessels of the upper extremities which are highly suggestive of, if not indistinguishable from, Raynaud's disease.

In thrombo-angiitis obliterans the pathological picture is that of a chronic inflammation of the arteries and veins, mainly of the extremities, with a tendency to thrombosis. This depends on the duration and severity of the disease, and varies from slight thickening of the vessel wall to complete occlusion of the lumen by an organized thrombus with 'stiffening' of the wall. In the end-stages the arteries and veins and the accompanying nerves are usually bound up together by fibrous tissue consequent on proliferative changes. Microscopically there is infiltration of the adventitia, media, and intima, together with proliferative changes in all three layers, resulting in gross thickening of the vessel.

SYMPTOMS.

The symptoms of Buerger's disease are as follows: (1) Absence of arterial pulsation. (2) Claudication, trophic changes of the skin and nails, decreased skin temperature, and atrophy of muscles—all of which are the result of insufficient blood-supply. (3) Attacks of acute superficial phlebitis (in one-half of the cases). (4) Changes in colour of the extremities—pallor of the skin on elevation, and rubor on dependency of the extremity. The skin of the extremities is usually cold and red, which is the result of the contraction or spasm of the arterioles together with dilatation of the capillaries. (5) Rest pain, i. e., pain occurring in the absence of exercise. (6) Slow progression of the disease, frequently affecting first one extremity and then the other. (7) Acute ascending thrombosis of the artery occasionally. (8) Neuritic pains which occur as a result of ischaemia of the accompanying nerves.

In addition to the symptoms resulting from thrombosis of the vessels which have been outlined above, the vasospastic contraction of both the

diseased vessels and of the normal collateral vessels damages the remaining circulation. This observation is of prime importance in the treatment of the disease. Oedema of the lower extremities may complicate the clinical picture. X-rays of the peripheral arteries are usually negative, but in cases of doubt one can affirm the arterial occlusion by means of arteriography (*Fig. 384*). This procedure is one of academic interest rather than of clinical importance, as palpation of the vessels readily determines the presence or absence of pulsation.

Part of the impaired blood circulation, as has been noted above, is due to vasoconstriction of the arteries and arterioles. This must be eliminated and give place to vasodilatation in order to determine to what degree the interruption of vasoconstrictor impulses will result in a permanently improved circulation (*Fig. 385*). This can be done by the intravenous injection of non-specific foreign protein in the form of triple typhoid vaccine, as a result of which, during the fever reaction, all the arteries which are not occluded by thrombosis are opened to their maximum extent. Spinal anæsthesia produces the same effect, but is not usually employed for this purpose. The improvement in the circulation from the increased blood-flow can be measured by determining the difference in the skin temperature before and after injection of typhoid vaccine, the initial dose of which is usually 35 million. These values are formulated by Brown in the so-called vasomotor index. This is



FIG. 384.—Arteriogram of hand; showing absence of filling in many of the digital arteries and increased collateral circulation along the ulnar side of the hand.

of great value in prognosticating the benefits that can be expected to result from sympathetic neurectomy. The index is obtained as follows: The skin temperature of the extremity and the body temperature of the patient are determined by the constant room temperature; following the injection of vaccine, an increase of the skin temperature and of the mouth temperature occurs. The temperature changes of the separate digits of all the extremities are measured by the thermocouple. Hourly readings are made until the maximal rise of fever has been obtained. The difference between the reading

of the skin and mouth temperature gives the indication for the existence of vasoconstriction. Thus the rise of the mouth temperature may be $2^{\circ}\text{C}.$; the rise of skin temperature in one digit may be $8^{\circ}\text{C}.$; the difference is 6, and this, divided by the rise in mouth temperature (2), gives a reading of 3—the vasomotor index. The range of increased skin temperature of the extremity alone, e.g., from $25^{\circ}\text{C}.$ to $35^{\circ}\text{C}.$, indicates the amount of heat elimination resulting from vasodilatation, or, in other words, the increase in the peripheral circulation.

In cases of arteriosclerosis little change in the peripheral skin temperature can be demonstrated. This is also true of those cases of thrombo-angiitis



FIG. 385.—Colour changes in the feet in a case of thrombo-angiitis obliterans before and after sympathectomy.

obliterans in which most of the vessels are occluded as a result of organic changes; but in those cases in which the impaired circulation is in some measure caused by spasm of the collateral arteries the peripheral temperature will increase considerably. It may be mentioned that there are certain contra-indications for the administration of typhoid vaccine. These are: (1) Active tuberculosis; (2) Advanced arteriosclerosis, on account of the possible danger of producing myocardial thrombosis (P. S. Hench). For this reason vaccine is seldom given to patients who are more than 50 years of age.

TREATMENT.

Treatment of this condition depends on the stage and severity of the disease. Patients who show only the symptoms of claudication should be treated by a medical régime. This includes limitation of exercise, protection from trauma and cold, contrast baths, the so-called Buerger exercises (the foot elevated for one minute to 90° , held dependent for one minute, and then raised to the horizontal level for one minute). Dry heat is also of value. When the disease is progressive, rest in bed is essential together with the other forms of treatment. Vasodilatation produced by artificial fever may be used

to give temporary relief. Intravenous injection of triple typhoid vaccine, as used for the cutaneous temperature test, first of 25 million, then increasing the dose by 25 million twice a week, produces a moderate fever reaction which is accompanied by vasodilatation, and is the method that is most commonly used. Other ways of obtaining a temporary vasodilatation are intramuscular injections of sulphur, and acetylcholine. These are less effective than the injection of typhoid vaccine. Prompt but short-lasting vasodilatation can be obtained by giving alcohol by mouth. The results obtained by the injection of tissue extracts are still uncertain. Insulin-free pancreatic extracts are said to cause some vasodilatation, but experiments at the Mayo Clinic have shown that, if present at all, this reaction is only slight and evanescent. However, these extracts seem to have some beneficial influence on the disabling symptoms of claudication.

Patients in whom no pulsations of the peripheral vessels can be felt should be warned not to undergo any surgical operation, however trivial, on the extremities because superficial infections and small wounds—for example, in an operation for ingrowing toenail—heal very slowly on account of the inadequate blood-supply and may cause gangrene which finally necessitates amputation.

The results of medical treatment are far from ideal, though the number of amputations which have to be performed for this disease has been reduced from 25 per cent in untreated cases to 14 per cent in cases which have received adequate medical treatment, and to 5 per cent in those who have had sympathetic neurectomy performed in addition.

The beneficial effects of operation are due to the elimination of vasoconstrictor impulses to the peripheral vessels. This is accomplished by resection of the lumbar sympathetic ganglia with the intervening trunks when the lower extremities are involved. The second lumbar ganglion is usually the last ganglion to receive preganglionic fibres, the white rami communicantes, but in order to eliminate the possibility of rami reaching the sympathetic trunk at a lower level and conveying vasoconstrictor impulses, the third and fourth lumbar ganglia are always resected in addition. Access is obtained by a mid-line abdominal incision and incisions in the posterior peritoneum on each side. The details of the surgical technique need not be described more fully here. (*Figs. 386, 387.*)

In order to obtain a complete vasodilatation of the upper extremities, bilateral resection of the cervico-thoracic ganglia with the intervening trunk is performed. The posterior mid-line approach is used, with resection of a portion of the first rib and the transverse process of the first thoracic vertebra on each side. The inferior cervical and first thoracic ganglia with the upper portion of the thoracic trunk are removed. Usually the second thoracic ganglion can be removed through the same approach; all the ascending rami to the first thoracic and eighth cervical nerves should be divided (*Figs. 388-390*). The neurosurgeons at the Mayo Clinic believe this operation to be more thorough than the antero-superior approach, as the latter renders more difficult the removal of all the rami communicantes arising from the first and second thoracic ganglia.

As the sweat glands and sebaceous glands are innervated by fibres which are intermingled with the sympathetic vasoconstrictor nerves, the skin of

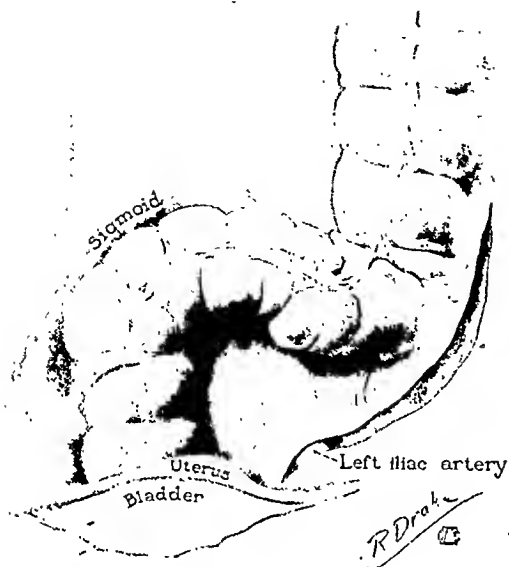
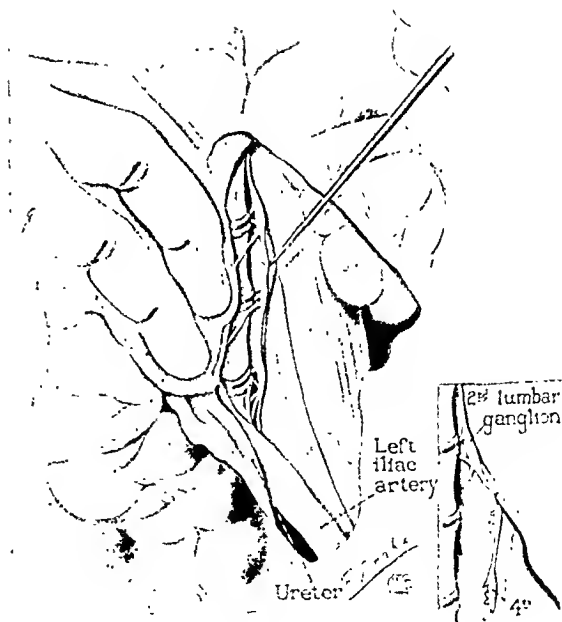


FIG. 386.—The incision in the lower left posterior portion of the peritoneum, permitting elevation and retraction of the sigmoid and descending colon medially.

FIG. 387.—Exposure and resection of the left lumbar sympathetic trunk, with the second, third, and fourth lumbar ganglia.



the extremity after the operation becomes quite dry. Complete absence of sweating indicates that the operation has been complete; if present, the operation has been incomplete. In order to determine this point, the patient is placed in an electric 'baker', and strips of cobalt-blue paper are placed on the skin. In areas where there is sweating the colour of the paper changes to pink. Although post-ganglionic fibres to the hypogastric plexus are cut in the lumbar sympathectomy operation, sphincter disturbances never occur. Cervico-thoracic sympathectomy produces a Horner's syndrome, which, however, is hardly noticeable, and causes no disfigurement when a bilateral operation has been performed. It is interesting to observe that following a complete sympathetic neurectomy the extremities become warm, but no redness of the skin occurs. This indicates a dilatation of the arteries and arterioles, but not of the capillaries. This observation is confirmed by the fact that under the capillary microscope the capillaries are not dilated, but that the rate of the blood-flow is increased.

Selection of Cases.—The indications for the operation of sympathetic neurectomy are: (1) In those cases in which the vasomotor index is sufficiently high, or in which there is an adequate range of increased temperature in the extremities following the administration of typhoid vaccine. (2) In progressive cases in which unsatisfactory results are obtained by medical treatment. The contra-indications may be summarized briefly as follows: (1) The presence of acute fulminating ulceration. (2) Progressive gangrene. (3) Low vasomotor index, as obtained by the skin temperature tests. Brown states that unless the rise of temperature in the skin is at least twice as great as the rise in mouth temperature, operation is contra-indicated. In this connection Mayo and Adson also state, "It is obvious that vasodilatation cannot be produced in an arterio-sclerotic or occluded artery and that surgical intervention is useless unless there is positive evidence of vasospasm in the remaining non-occluded arteries". (4) General contra-indications such as debility, cachexia, etc.

It is impossible to give more definite rules regarding the advisability of operation, as each offers an individual problem. It is well to hospitalize the patient a few weeks before operation in order to try to improve his circulation by a medical régime. Patients who present themselves with acute lesions, such as ulcers or gangrene, are not operated on until healing of the ulcer begins,

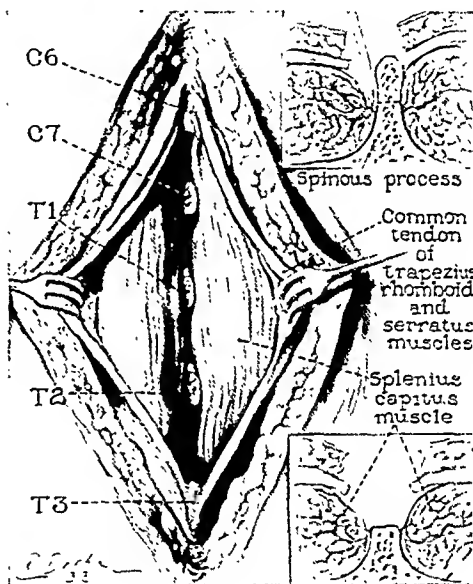


FIG. 388.—The dorsal mid-line incision with reflection of the common aponeurotic tendon of the trapezius, rhomboids, and serratus posterior muscles and resection of the spinous processes.

and in gangrenous digits until the line of demarcation between healthy and diseased tissue appears.

When sympathetic neurectomy is held to be contra-indicated amputation may have to be resorted to in certain cases. The most frequent indications for amputation are: (1) Progressive gangrene; (2) Uncontrollable pain in the presence of extensive occlusion of vessels which do not respond to vasodilating agents. It is often difficult to decide at what level to amputate. Occasionally an amputation of a toe or finger for a small area of superficial gangrene is successful, but usually a high amputation is indicated. If the popliteal artery is felt on examination to pulsate, amputation below the knee may be tried, but even then, in many cases, re-amputation above the knee

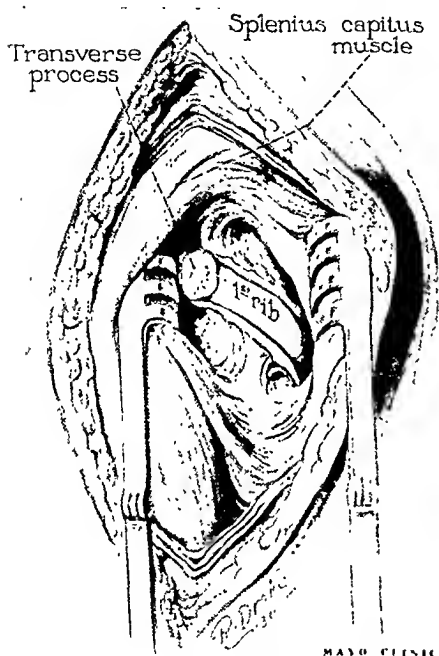


FIG. 389.—Exposure of first rib and transverse process of the first thoracic vertebra preliminary to resection.

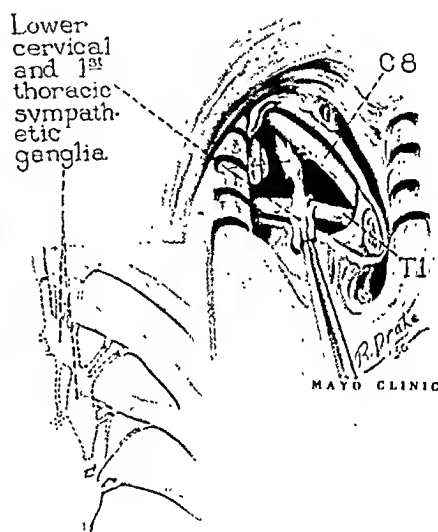


FIG. 390.—Resection of the lower cervical and first thoracic sympathetic ganglia with the trunk, and of rami extending from the second or third thoracic ganglia to the first thoracic and eighth cervical nerves.

will be necessary as the result of poor healing. Occasionally the amputation is complicated by gas-gangrene infection, which, however, is usually fairly benign.

Results.—A recent review of 100 consecutive cases of thrombo-angiitis obliterans showed that bilateral lumbar sympathetic ganglionectomy was done 89 times and cervico-thoracic sympathetic ganglionectomy 15 times. Four patients were operated on for the disease affecting both upper and lower extremities, which accounts for the 104 operations. There were 96 men and 4 women; the average age on admission was 35 years. Eighty-seven patients were markedly improved, with relief of pain and claudication assessed at an

average of 80 per cent, and improvement in the healing of ulcerating lesions of the skin. In the absence of advanced trophic lesions sympathectomy checked the progress of the disease in the opposite extremity in every case.

RAYNAUD'S DISEASE.

The main features of Raynaud's disease are so familiar that only a brief recapitulation is necessary. The disease should be differentiated from organic diseases of blood-vessels, for no organic changes in the vessels can be detected. There is, however, some vasomotor disturbance. Brown calls the condition a vasomotor neurosis of a spastic type. As a result of this disturbance, there are phases of symmetrical colour changes of the skin of the extremities, and sometimes of the nose and ears, associated with more or less severe pain. The arterial pulsations are present.

The disease is seen almost exclusively (about 80 per cent) in women, usually of the asthenic nervous type, and of a comparatively young age (average 31 years). The attacks are mostly precipitated by cold and exposure, resulting in contraction of the small arterioles, capillaries, and venules. When the skin in the acute phase is observed under the capillary microscope, only a small number of partially filled capillaries are seen. After a few moments, marked cyanosis of the extremities occurs. The stage of cyanosis is due to partial relaxation of the venules with a back-flow of blood in the capillary loops. During the last stage of the attack, increase of the temperature occurs spontaneously as a result of increased circulation following the opening of the capillaries, and the colour of the skin changes to red. All stages are seen between the almost normal response of the vessels to vasoconstrictor impulses induced by exposure to cold and the more severe attacks which follow only slight temperature changes, and in which there may be such complications as œdema of the extremities, trophic acral changes, dry ulcers, scleroderma, and occasionally gangrene.

The cause of the disease is unknown, but it is believed that the condition is of central origin in which there is an abnormal response on the part of the vasomotor centre, and that the local changes in the extremities are mediated through the vasomotor fibres. Krogh and Lewis, on the other hand, hold that the phenomena are due to a local cause associated with an abnormality digital arteries and arterioles, resulting in defective circulation.

TREATMENT.

The treatment of Raynaud's disease is dependent on the severity of the symptoms. In mild cases good results are obtained by eliminating the predisposing factors, e.g., by wearing warmer clothing and living in a warm, dry climate; but not all cases can be helped by these measures, and the severity of the symptoms together with the degree of disability may justify an operation. Cases have been reported in which temporary improvement was seen following the injection of typhoid vaccine.

The surgical treatment is essentially the same as that already described for thrombo-angiitis obliterans. As Raynaud's disease attacks the upper

extremities more frequently than the lower, it follows that cervico-thoracic sympathetic ganglionectomy and trunk resection is the usual operation. In a series of 54 cases, it was employed 39 times, while lumbar sympathetic ganglionectomy was employed 15 times. In the former group the average relief of pain and of colour changes in uncomplicated cases was 85 per cent, and in the latter group 100 per cent.

The incomplete results which are sometimes seen following operation on the upper extremities are apparently due to failure to resect all the vasomotor fibres. The more complete operation, as now advocated and practised, by including the second thoracic ganglion, the cervico-thoracic trunk, and all the rami going to the eighth cervical and to the first and second thoracic nerves, obviates the previous criticism and enhances the prognosis.

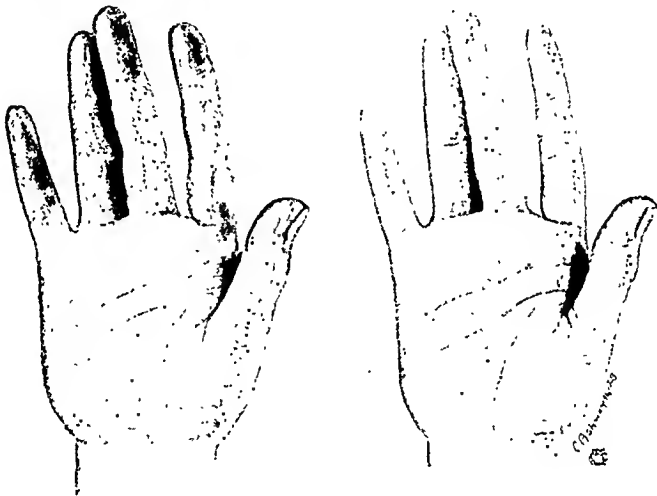


FIG. 391.—Colour changes in the hands in a case of Raynaud's disease before and after sympathectomy.

Therefore it can be said confidently that as a result of sympathetic neurectomy in Raynaud's disease the pain is relieved, abnormal colour reactions disappear, the skin of the extremities becomes pink in colour, dry, and warm, with a sustained increase in surface temperature. As Adson and Brown have stated, "The striking, maintained, and unequivocal therapeutic effects of lumbar and dorsal sympathetic ganglionectomy in Raynaud's disease seem to warrant the belief that surgical control in this disease is an accomplished fact." (*Fig. 391.*)

CASE REPORTS.

The following two cases are of interest in that they represent the extremes of the age-incidence group and were admitted consecutively at the clinic. The first was a woman, aged 52, and the second a girl, aged 11.

Both these patients had a bilateral cervico-thoracic sympathetic ganglionectomy performed, and the girl had in addition a bilateral lumbar sympathetic ganglionectomy.

Case 1.—Mrs. J. J., aged 52, was admitted to the clinic on Dec. 6, 1932. Her chief complaint was "numbness of the fingers and nervousness". She stated that she had suffered from many nervous breakdowns and gave way to frequent spells of crying. This nervous instability was made worse six years ago by the death of her husband, by her daughter's going blind, and by her mother's becoming insane. It was still further intensified by worrying over her present condition. Her present emotional stress amounted almost to an anxiety neurosis. Having become engrossed in the periodic attacks of numbness and colour changes that affected her fingers and toes, she related that she had spent much time in public libraries reading all the medical literature available, and that she had finally made her own diagnosis of Raynaud's disease. She then consulted forty-three doctors. They all stressed the nervous element in her condition and advised no active treatment. She remained unsatisfied, and, fortified by the comforting reports she had read of successful surgical treatment in other cases, she presented herself at the clinic with the firm intention of undergoing operation.

She had had numerous operations in the past: appendicectomy followed by peritonitis in 1910 and subsequently right salpingo-oophorectomy, complicated by intestinal obstruction; drainage of a kidney abscess; tonsillectomy; and left femoral perivascular sympathectomy ten months ago. This last operation gave her no relief.

ON EXAMINATION.—The patient was an obese (weight 188 lb.), well-developed elderly woman. Inquiry revealed that numbness and changes in colour in her fingers were first noticed sixteen years ago. These changes were brought on by cold and damp, and went through two phases, red and white. Several years later the toes were similarly involved. There was no pain at first. For the last three years the changes occurred in spasms which became progressively severe: in the fingers they were accompanied by pain, whereas in the toes there was numbness only. There was no constant stimulus, but the spasms were initiated by using the fingers as in sewing, particularly in damp weather and when exposed to the cold. The painful spasms in the fingers, according to the patient, formed a major disability, and she repeated her assertion many times that she would not rest until operation was performed. The numbness in her toes was unpleasant but not a disability. There were no trophic changes, ulceration, scleroderma, or arthritis.

The physical examination was otherwise negative, except for varicose veins in the right leg and slight loss of all types of sensation in the tips of the fingers and toes. Fundus examination showed a slight degree of sclerosis of the retinal arteries with structurally full discs; the eyes were otherwise negative. Urinalysis was negative except for slight albuminuria. Haematology: erythrocytes 4.54 million, leucocytes 6900, haemoglobin 97 per cent, blood-sugar 105 mgrm., B.P. 128/90, pulse 80. Basal metabolic rate + 2. Flocculation tests were negative for syphilis. X-rays of the chest, spine, and extremities were negative. More detailed examination of the extremities showed that there was a two-phase colour reaction involving the fingers and toes. On elevation there was pallor associated with numbness, and on dependency there was rubor associated with tingling. The arterial pulsations were all normal, and there was no clinical evidence of organic obstruction or arteriosclerosis.

The cutaneous temperature tests and vasomotor studies were not completed in this case owing to the emotional distress that they produced. The consultant's note states, "Patient gets too emotionally upset to conclude any satisfactory studies regarding vasospasm of the hands. She says there has been no change since the tissue-extract was given; she says the hands feel as if they would change in colour if she went out, but this upsets her for the whole day and makes her so nervous, and gives her so much pain, that she cries even thinking about it. Therefore I would suggest discontinuing all studies meantime."

Operation was then advised because: (1) the condition was progressive but uncomplicated, (2) the pain during the spasms was intolerable, (3) the result should be good. The patient's age and emotional instability, in which the main factors were environmental, were not considered of themselves to be any contra-indication.

OPERATION.—On Dec. 15, 1932, Dr. Adson performed a bilateral cervico-thoracic sympathetic ganglionectomy and trunk resection under ether anaesthesia. The surgical card reads: "Approach through a mid-line dorsal incision. The common tendon of the trapezius, rhomboids, and the serratus posterior was elevated and subsequently closed after removal of the spinous processes of the first and second dorsal vertebrae. Entrance made through the first rib on each side. The ganglia were all about the usual size. The lower cervical ganglion on the left side presented a very beautiful stellate arrangement and communicated with the first thoracic by a broad trunk. The operation was comparatively easy, and there was no loss of blood. Prognosis should be good. Mortality less than 0.5 per cent".



FIG. 392.—Case 1. The wound post-operatively, showing the extent of the incision.

SUBSEQUENT PROGRESS.—Convalescence was satisfactory and the wound healed by primary intention (*Fig. 392*). The patient complained of indefinite pains in her joints and a little pain in the region of the right antecubital fossa. Twenty-five days after operation a sweating test showed that resection of sympathetic fibres had been complete. In this case there was no sweating from the shoulders to the finger-tips; elsewhere over the body sweating was profuse. There was a good gain in surface temperature as studied under normal room-temperature conditions. There were no colour changes in the hands when immersed in cold water. At the time of her dismissal on Jan. 11, 1933, the hands were dry and warm and it was clear that the circulation had been greatly improved.

On Jan. 19, her home doctor stated in a letter: "I was very glad to see her again and am much impressed by the good results of the operation". On March 18, the patient wrote as follows, "My back is doing nicely though it tires easily.

The circulation in my hands is practically very good. . . . I truly believe my whole nervous system has undergone a change. The tension has gone and I seem to be calm, cool and collected. . . . You know how happy I am."

Case 2.—L. M., a female, aged 11, was admitted to the clinic on Jan. 20. 1933. She complained of "all her fingers and toes turning dark blue every time they become the least bit cold". Her family history was negative, although her mother was said to be "very nervous". The patient had tonsillectomy performed three years ago; there were no other previous diseases or injuries.

ON EXAMINATION.—She was a well-developed but rather thin little girl (weight 80 lb.), timid and shy on examination, but essentially of a rather unemotional and stolid type.

Her symptoms began about one year previously. At that time she had attacks of cyanosis and numbness of the fingers and toes, induced by exposure to cold. One month previous to admission she had noticed a small ulcer at the tip of her right index finger. The symptoms were practically absent during this last summer, but with the coming of autumn they re-appeared, and during this winter became much worse than ever before. She wore heavy gloves, but even then the fingers became very blue and cold; they also became swollen. There was no pain or tingling associated with the attacks, but a sensation of numbness. She complained of her fingers more than her toes. Her physical examination was essentially negative. There was some dental caries and a slight enlargement of the cervical glands. Urinalysis was negative. *Hæmatology*: erythrocytes 4.29 million, leucocytes 6900, sedimentation index 2.3, B.P. 120/60, pulse 72. Flocculation tests were negative. Eye examination was negative except for mild structural blurring of each disc. The X-ray examination of the chest, hands, and feet was negative. The central nervous system was negative.

The fingers and toes were blue and cold. The index and middle fingers of the right hand were swollen. The ulcer on the tip of the index finger was close under the nail; it measured about 7 mm. in diameter and was surrounded by concentric layers of thin desquamated skin. There was beginning desquamation at the tip of the middle finger but no actual ulceration. The cold test was negative for hypertension or for a pre-hypertension state.

On Jan. 26, 1933, the vasomotor test resulted as follows (the readings in Centigrade):—

DIGIT		INITIAL TEMPERATURE	MANDUCH TEMPERATURE	RANGE OF TEMPERATURE	RISE IN MOUTH TEMPERATURE	INDEX
Right 2 F.	..	25.5°	32.8°	7.3°	1.1°	5.6
Left 2 F.	..	27.0°	34.7°	7.7°	—	6.6
Right 1 T.	..	24.1°	35.6°	11.5°	—	9.4
Left 1 T.	..	24.0°	36.5°	12.5°	—	10.3

Conclusion: "Good vasodilation; adequate for operation."

Thus a diagnosis was made of Raynaud's disease involving both upper and lower extremities, with secondary thrombosis and occlusion of the digital arteries, especially that of the right index finger.

FIRST OPERATION.—On Jan. 31, 1933, Dr. Adson performed a bilateral cervico-thoracic sympathetic ganglionectomy and trunk resection under ether anaesthesia. Approach was made by the usual method with resection of a portion of the first rib on each side. The ganglia were normal in size and distribution.

COURSE.—The patient's convalescence following this operation was entirely uneventful. After the vasodilating effects of the anaesthetic had worn off and could be discounted the hands remained persistently warm, dry, and pink. The small trophic lesions on the tips of the middle and index fingers of the right hand began to heal immediately. There was a definite Horner's syndrome on each side. On March 8, thirty-seven days after operation, a sweating test showed that a complete operation had been performed, with interruption of all the vasomotor fibres to all the sweat glands of the upper extremities (*Fig. 393*).

SECOND OPERATION.—On Feb. 20, twenty-one days after the previous operation, the patient had convalesced sufficiently well to permit of the second operation for the relief of the disease affecting the lower extremities. Consequently a bilateral lumbar sympathetic ganglionectomy and trunk resection was carried out under ether anaesthesia by Dr. Adson. The usual transperitoneal approach was used, with incisions lateral to the caecum and to the pelvic colon in turn. The abdominal and pelvic viscera were normal. The ganglia were easily exposed, and the second, third, and fourth with the intervening trunk were removed on each side. They were of the usual size, shape, and distribution. The wound was closed in the usual manner.

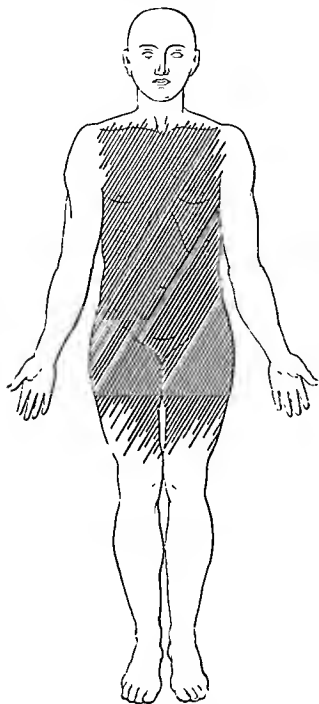


FIG. 393.—Case 2. Diagram illustrating the result of operation following the sweating test: the absence of sweating is indicated by the non-shaded areas.

SUBSEQUENT PROGRESS.—Following the second operation the patient's convalescence was again without incident. The feet and toes became warm and dry and "very comfortable". There were no symptoms or changes in the normal function of the bladder and bowel. On March 7, fifteen days later, the skin temperature of the hands and feet under normal room conditions was determined by hourly readings for six hours. The results were as follows:—

Right foot	32.6° C. (aver.)
Left foot	32.4° C. (")
Right hand (dorsum)	31.8° C. (")
Left hand (dorsum)	32.8° C. (")

This test showed that an average high level of surface temperature was maintained, and indicated that a definite and persistent increase in the peripheral circulation had been accomplished.

On March 11, nineteen days after the second operation, the patient was allowed to return home. The post-operative convalescence record, in summing up stated, "There was a striking improvement in the condition of the hands and the feet. They were warm and dry, and all her symptoms were entirely relieved."

This communication is a brief outline of the views held and the treatment employed at the Mayo Clinic in regard to certain vascular diseases which I have had a special opportunity of studying during the tenure of a Rockefeller Travelling Fellowship. While it is realized that many aspects of these diseases are familiar to those who are engaged in work in this field elsewhere, it seemed that the above descriptions might be of interest to others who are not engaged in a special study of the subject.

My thanks are due in particular to Dr. A. W. Adson, for whom I had the privilege of acting as First Assistant, for his permission to make full use of his personal statements and publications.

THE SELENIDE TREATMENT OF CANCER.

By A. T. TODD,

ASSISTANT PHYSICIAN, BRISTOL ROYAL INFIRMARY.

I COMMENCED active research on the treatment of cancer about 1926; at that time treatment by what was called 'colloidal lead' was much to the fore. As I did not wish simply to repeat the work of others, I decided to try to combine the possible action of lead with that of selenium. Selenium was first used on a scientific basis by von Wassermann,¹ and on one occasion the results were promising. It appeared possible that the combination of two agents might be more effective than either singly. Therefore I had lead selenide made in colloidal form, and after careful toxicology experiments on animals, employed it in cases of guaranteed inoperable cancer.

This selenide, called 'D4S', has been on the market for five years. My results from its use were good, all things considered. Of the cases treated, 10 per cent can be claimed as four-year cures. About 50 per cent of the cases showed marked prolongation of life, with comfort as a rule. Its analgesic effect is often marked, giving relief when morphine and other analgesics fail. But this system of treatment was slow and required much care on the part of the practitioner and much co-operation from the patient; in addition, as in all chronic diseases, it requires that the practitioner shall convey his real, or assumed, belief in the efficacy of the treatment. Care in dosage is essential: too small dosage gives no result; too large dosage hastens the growth of the neoplasm. I still maintain that this system¹ should be employed as a routine for a year after presumed complete operation; in view of my results it appears highly probable that a reduction in the number of recurrences would follow. But from now onwards the colloid R.A.S. should be substituted for the old D4S.

Certain of my cases failed to benefit from this selenide, completely or in part. I determined, therefore, to try if its action could be augmented by concurrent radiation from X rays and radium. The cases were treated cautiously and never received more than half the total dosage which would have been given to a case treated exclusively with radiation. The result was disappointing at first sight, and certainly surprising; on applying radiation in the usual dosage to cases impregnated with lead selenide there always followed a striking and rapid intensification of the growth of the neoplasm: cases which had been stationary for months would die within a few weeks of acute cancerous invasion. This happened in twelve consecutive cases. Concurrent radiation was stopped in man and repeated on mice with implanted Twort carcinoma, with the same result—radiation on a mouse impregnated with lead selenide caused intensification of the rate of growth of the implant.²

The significance of this finding was not grasped at first, for a good deal of experimental work was necessary. It was found that massive dosage of D4S—that is, the largest doses tolerable by man or animal—gave the same response, a marked and prompt acceleration of neoplastic growth. Possible explanations were: (1) That radiation had in some way intensified the action of the colloid and so converted a small into a large dose: from the work of others and ourselves² this result must be due to the selenium, for radiation after lead colloids free from selenium does not give this result. Or (2) That the presence of the selenide had converted a relatively small into a relatively large dose of radiation; for my experience is that, with the exception of a few types of specially sensitive growths, intensive radiation also has a stimulating action on neoplastic growth.

The second of these hypotheses appeared to be the more attractive, and steps were taken to make another selenium colloid which would be more amenable to combined colloid-radiation therapy. We found that by reducing selenium dioxide in the presence of my sulphur colloid, selenium was taken up and a double colloid of sulphur and selenium resulted. We call this new colloid 'SSe', and it will be liberated for general use on publication of this article.

To appear to digress for a while. It may be asked: Why so much talk about colloids? To my mind the disease cancer is closely related to the granulomata, the connecting link being lymphadenoma. Partial proof has been afforded recently that the latter disease is due to a virus. I had become convinced long before this that cancer was an infective disease and probably due to growth stimulation from the presence of a virus. I admit that final proof is not yet attained, but if the evidence is weighed without bias, there is little ease for any other conception. Space does not permit me to go into this question more fully. I considered, then, that cancer was an infective disease and that a defence mechanism of mesoblastic tissues was present, but that this defence always failed. This tissue I called 'junction tissue', and I gave some proof that chronicity and radio-sensitivity of a cancer were, as a rule, proportional to the amount of this junction tissue.³ It consists of fibroblasts, lymphocytes, plasma cells, eosinophils, basophils, and macrophage cells. From our knowledge of defence mechanisms in other diseases we should regard the macrophages as the key cells, as they appear to govern the function of the others. These macrophages can only be influenced by colloids; they engulf any electro-negative colloidal particle. If the particle is incapable of being broken down by the enzymes of the macrophage, it is simply stored or passed away into the lymph channels. But if the particle can be chemically altered by the cell enzymes, there are several possibilities: some innocuous compound may result; a substance poisonous to the cell which has produced it may be formed; a compound which stimulates the metabolism of the cell may result. Thus a colloid is essential, and intravenous administration will be necessary for certain access to the macrophage system.

In brief, then, cancer is regarded as an infective disease with a defence mechanism; a system of treatment which should increase this defence was attempted; if stimulation of the defence then became adequate, the growth

of the neoplasm should be inhibited. That is, the action of the treatment in cancer, as in the other chronic diseases, should be in assisting the process of natural cure, for direct action, to try to poison the disease tissue itself, has completely failed.

This hypothesis demands proof, and we can give a certain amount. Radiation alone increases the amount of junction tissue, macrophage included, if correct dosage is employed. Lead selenide alone, and lead selenide after radiation, give the same result, and with increase of junction tissue there is atrophy of the neoplastic tissue. But radiation of the same dosage after treatment with lead selenide caused a poverty of junction tissue, usually disappearance of macrophages, and an increased rate of growth of the implant.³

The newer method of treatment consists of radiation plus intravenous injection of selenium colloids. Have we any sort of proof that radiation can act upon and alter the selenium particle? There is a good deal of proof existent. The alteration in the conductivity of selenium from radiation of light rays is the basis of the action of the selenium cell, which is widely used in sound films, burglar-traps, some traffic signals, and television. It was known that X rays and gamma rays also produced a change; this change is called 'ionization' by the physicists, and for this reason I have called the new method the 'ionization method'. I do not specifically claim that such ionization is the cause of the results obtained, but that it may be the cause. I do claim that some change follows the application of deep X and gamma rays to a tissue impregnated with this new selenium colloid, and we have collected a certain amount of proof.

1. *Physical*.—Radiation by sunlight flocculates the colloid and the selenium migrates towards the radiation. On pure selenium, the selenium bridge, we find that X rays produce a rapid increase of conductivity; heat rays have the opposite effect, but less rapid. Radium rays slowly and steadily increase conductivity up to twenty-four hours, the time-limit of our experiments. We have also tested the effect of the shorter Hertzian waves, those with a wave-length of about 4 metres. Here I should like to thank Mareoni Company Ltd. for their generosity in lending us the necessary apparatus. On tuning the external circuit we find a sharp increase of conductivity for a short period, then a steady diminution—the latter being due to the heat effect, but tuning and detuning still show smartly. A selenium colloid was prepared as free from electrolytes as possible; this has also been tested against the same types of radiation; X rays, radium rays, and Hertzian rays all show a diminution of conductivity. This contradiction is only apparent, for the current is carried by the electrolytes and not the colloidal particles; the latter are ionized, however, and then adsorb electrolyte and thus diminish conductivity. The selenium colloid which we use does not show this effect, for there is so much available electrolyte that any ionization effect is masked *in vitro*. A sulphur and a tellurium colloid do not give these results.

2. *Biological*.—The evidence on mice has been noted; radium radiation after impregnation with lead selenide sol regularly produced acceleration of neoplastic growth and atrophy of junction tissue. A long series of experiments on radio-sensitive protozoa has been carried out. To cultures of the

protozoa various colloids were added and the result of radium radiation observed. In the presence of suitable amounts of selenium sols, radiation steadily increased the death-rate as compared with non-radiated cultures. Sulphur sols had the opposite action. A tellurium sol had no effect.

3. *Clinical*.—The clinical proof is, in the main, the success or otherwise of the system of treatment, and I think the results will speak for that. Patients are only accepted into my clinic after they have been given up by surgeon, radiologist, and radium officer. Most of them have already been treated by their methods before I take them, and treatment has, of course, failed. All the cases are advanced, and fully a fifth can be regarded as moribund. It is quite often found that a patient has died before a bed has become available, although cancer cases are given priority. I think it will be conceded that a high rate of two- to four-year cures is not to be expected in such material. Until quite recently no selection of cases has been made; if there was room for the patient, then the patient was taken.

That an action on the patient does follow the application of X-radiation will quickly become apparent to those who follow the system. After an intravenous injection of SSe there may be some trivial and evanescent effects such as cough or a short rigor; then, three to twelve hours later, true focal reaction sets in. This consists of intensification of the symptoms produced by the neoplasm, and, when superficial, it is frequently seen that congestion and some œdema have appeared around the growth. This focal reaction passes off in a longer or shorter time, and then there is usually freedom from pain and general euphoria. Two days later, this time to allow effective storage by the macrophage system, and to be well over the reaction produced by the colloid, the dose of deep X rays is applied. If the dose is adequate, focal reaction appears again, but with a much shorter latent period. If a non-selenium colloid is administered, there is no focal reaction and the same dose of X rays produces no result. This has been done on many occasions, in order to exclude any factor of suggestion. The same dose of X rays at a 12- to 16-day interval after colloid injection also produces no result. This finding was not expected; it is simply what has been found. As the only change in selenium on radiation is ionization, I call the method the 'ionization method'. Mr. C. Joll, in his recent Long Fox Lecture, criticized my system on account of this name; he stated that certain physicists had assured him that the amount of ionization reproduced from the dosages I use would be inadequate; I think they meant 'inadequate to kill the cancer', and there I agree, for no attempt is made to kill the cancer directly. I do not think they can deny that this trivial ionization might stimulate the macrophage system. Proof of ionization in the living subject is quite impossible.

Later it appeared to be desirable to determine whether a radio-active selenium colloid might be effective; if ionization does occur, then the taking up of such a colloid into the macrophage system in the junction tissue should allow feeble, but continuous, ionization at the site itself. Radium residues, consisting of radium G and traces of the higher breakdown products of disintegration, were converted into colloidal selenides in a manner similar to that employed for D4S. We call this newer colloid 'R.A.S.' It is very similar in its appearance and properties to D4S and is quite stable in the

absence of oxygen. It is feebly radio-active, but I have not the facilities to measure its exact intensity; it does slowly blacken a photographic plate protected by light-proof paper. Its toxicity to man and animals is very similar to that of D+S, but its local action in causing obliterative endophlebitis is much less marked. It was seldom possible to get more than an average of six intravenous injections in mice with D+S, but with R.A.S. weekly injections can be made almost indefinitely. The results on the slow-growing strain of Twort carcinoma we propagate are much better than those given by D+S; with the latter we averaged about 10 per cent of regressions, but with R.A.S. we now get a steady 25 per cent.

The parenteral administration of radium salts is highly dangerous; the results, mostly from the United States, show that some fatal disease is almost an invariable sequel—osteomalacia, anæmia, leukæmia, or osteogenetic sarcoma. R.A.S. has a very much more feeble radio-activity than any pure radium salt, and though it has been employed in man for two years, no ill effects have been observed. But as two years is a shorter time than is the latent period of the diseases noted, a number of animal experiments have been carried out. Mice and rabbits have been injected with as large doses as they would tolerate for a time, then observed for six months; this period corresponds to about twenty years man/mouse and seven years for man/rabbit. The animals remained well as far as could be seen. They were killed and examined histologically after death. No abnormalities were found.

The last new remedy in the system is Paré's ointment. Ambroise Paré treated malignant ulcers by filling the cavity with a sheet of lead cut to shape and rubbed over with mercury. He claimed that it was a useful treatment, and he was a very accurate observer. I considered that a much more powerful action would be likely from the use of finely powdered lead amalgam embodied in an ointment. I think that it is really a useful application for malignant ulcers, or threatened ulcers, except those so near the mouth that poisoning would be likely: it has an antiseptic action, probably from breaking off of mercury salts. Patients are to be warned of its highly poisonous quality if taken by the mouth, and are advised to wash thoroughly after applying it. Nurses are advised to wear gloves. No case of poisoning has occurred in the clinic. I have never employed it alone, so that its potency is uncertain. It is manufactured by British Drug Houses Ltd., just as are the two newer colloids, SSc and R.A.S.

I have been criticized for not liberating methods of manufacture wholly and fully. There are several reasons why I have not. Firstly the medical journals would not welcome a very long purely technical account of the methods, and a full description would necessitate a long article. Then these selenium colloids require great technical exactitude in manufacture if a non-toxic colloid is to result; trivial impurities, in distilled water, for instance, will produce a colloid of high toxicity. The firm selected for its high status was requested to send a chemist to the Bristol laboratories to be taught the method of manufacture; thus colloids exactly similar to those we use are produced. I feel certain that if this precaution had not been taken, firms would have produced colloidal preparations of differing properties from those

we used. They would have done so quite unknowingly, but then these findings would have been discredited.*

THE TECHNIQUE OF THE NEWER METHOD.

The patient is warned that treatment will be prolonged, extending over years, if the result is favourable; a weekly attendance will be necessary. If he cannot engage to do this, then he is not accepted for treatment. Of late the moron type has not been accepted, for co-operation is impossible. Carcinoma of the alimentary tract with hepatic metastases is only accepted if the primary is capable of removal, for while primary colon requires high dosage, hepatic metastasis requires low dosage. Other than this, no selection is made, and until recently no selection whatever was made. Every case has to be guaranteed untreatable and to be free of diagnostic doubt.

When surgical removal of a reasonably large part of the mass is regarded as a fairly safe procedure, the patient is referred back to the surgeon for what we call the 'toilet operation'; this is especially desirable in septic fungations, for sepsis causes false reaction and one is impeded in assessing true reactivity. Even in non-septic cases removal of as much as possible is desirable on first principles, as this leaves less for us to deal with. Such a toilet certainly improves results, but, breast cases excepted, it is not often possible. In a toilet operation difficult glands are not removed. In cases with rapidly-growing carcinoma, such as encephaloid of the breast, or sarcoma, one or two weeks of ionization are given before the toilet, with the object of getting some inhibition. Occasionally this preliminary has been so successful that toilet, or biopsy operation, has had to be put off altogether, as in the following case:—

Dav.—Sent to me by Professor Rendle Short. There was a large, rapidly growing lump springing from the pubis and extending upwards and backwards, involving the bladder, judging from the dysuria and frequency. The patient had lost much weight and become very weak. X rays showed a shadow less dense than bone but apparently containing some strands of ossification or calcification. The mass was about the size of a grape fruit. We decided to postpone biopsy until growth was stopped. On treatment rapid diminution in size took place and any interference was considered unjustified. The man has been discharged for two years and now is at work as a coal-hewer.

Next tests of blood and reactivity, purely for research purposes, are made, and then the patient is started on the medicinal and other treatment. As I have on several occasions given the rationale of the drug treatment,⁴ I will not repeat it; each patient takes $\frac{1}{16}$ gr. of dry thyroid extract once daily; three to six drops of radiostolcum daily; three doses of mist. calci thiosulphate (sol. calc. thiosulphate, 50 per cent, min. 40; glucose gr. 20; inf. caryoph. (1-7) $\frac{1}{2}$ fl. oz.; water to 1 fl. oz.; this replaces the older mist. calci chlorid. alb.) plus 5 gr. of sodium iodide after meals. If pain is experienced, we give aspirin, tab. amidopyrin co. (B.D.H.), allonal, or the other analgesics, morphine group excepted. As a last resort we give heroin, but

* No royalties whatsoever are paid directly or indirectly.

few cases respond unless its administration can soon be stopped. Many of the patients have been on large doses of morphine before they come to us; as a rule we are soon able to discontinue it. Pain usually lessens at an early stage of treatment; a case under treatment at the present time illustrates this:—

Cat.—Breast removed in Chicago: secondary deposits appeared in the lumbar spine and she was treated with some form of colloidal lead, probably the phosphate. Being aware of the diagnosis, she determined to come to England to see relatives before it was too late. She was treated at Cambridge and London by deep X-ray therapy, but went downhill. She was sent to my clinic, the surgeon reporting her condition as deplorable, as it really was. She had intense pain on movement and at rest; she vomited all she ate. Large doses of morphine gave little relief. Skiagrams showed large deposits in lumbar spine and ilium. She was treated by the method I am about to describe, and within a week was able to do without sedatives. Her pains disappeared and she was able to get up for a time. Later, mental blunting, transient diplopia, and choked disc indicated a cerebral metastasis: this is now being treated, the result at present being doubtful.

The tests noted below being completed, the radiologist is asked to give a time for treatment, and approximately forty-eight hours before this date an intravenous injection of SSe is given. The size of the initial dose varies with the size of the patient and the type of neoplasm. Sarcoma, rapidly-growing carcinoma, ovarian carcinoma, and hepatic growths are likely to be sensitive: an initial dose of 2.0 c.e. will probably be safe. Other types will start with 4.0 c.e. Reaction is awaited and its magnitude is noted. We try to avoid more than appreciable reaction; if much followed the first dose, the second dose will be reduced. If no reaction follows, then the dose is increased by 1.0 c.e. for sensitive, and 2.0 c.e. for non-sensitive, cases. This is repeated each week till reaction is obtained; but once the reactive dose is found, that dose is continued till reaction tends to wane, when again an increase is made. Pyrexial conditions, such as colds, increase reaction; if present, we make it a rule to halve the size of the injection.

After forty-eight hours the patient is radiated, and the first treatment is over the main mass, or, if pain is present, over the painful part. The dose given is $1\frac{1}{2}$ Holtznecht units, distance 30 in., 180 K.V., screen 0.3 mm. zinc. This dose is repeated weekly until 12 H. units have been given; but in a case not responding, we sometimes continue up to 20 H. units before stopping. After each application X-ray reaction is looked for; if it is marked, the dose of X rays is reduced the next time; if not present, and the colloid reaction was adequate, the X-ray dose is increased. If X-ray reaction is slight and colloid reaction trivial, then the colloid dose is increased first and X-ray dose increased later, if reaction is still absent.

When the first ionization course is over the patient begins alternation of colloids. He attends weekly and is questioned about symptoms, etc., and is given new supplies of medicines. For the first three weeks he is given R.A.S., the first dose being about two-thirds the amount of his last dose of SSe. If marked reaction follows, the second is lessened. Always just appreciable reaction is aimed at. After this three weeks, he has alternately one week SSe, the next R.A.S. For pyrexial interludes the dose of SSe is halved, but if it is a week for R.A.S. we substitute SSe instead. This alternation is to permit a slow, progressive ionization from the radiation of the R.A.S.

If progress is in doubt, or signs of extending growth appear, he is again started on ionization from X rays, but we do not like to do this within three months from the termination of the last course. If progress appears to be satisfactory, alternation is continued for at least six months, when a holiday from injections is given for about eight to ten weeks. After this, if progress is satisfactory, alternation continues; if unsatisfactory, ionization followed by alternation. When progress has continued satisfactory for about nine months, no colloids are given, the patient simply attends for observation and medicines. After six months of observation he is discharged, but told to attend at once if any suspicious symptom appears. So far no discharged case has ever returned with active disease of the type originally treated; one patient with mammary carcinoma did return after nine months of discharge with what appeared to be encephalitis. She died, and autopsy revealed primary carcinoma of the pituitary; no mammary growth was found.

Reaction.—It is important that a correct idea of what is meant by reaction should be obtained by practitioner and patient. Reaction means a definite, but moderate, aggravation of the symptoms produced spontaneously by the neoplasm. It may be severe if the initial dose is too great; it will tend to be severe if there is a coryza or similar pyrexial malady, but exclusive of these there should not be severe reaction. Severe reaction may be fatal; if not, it will probably make the patient avoid further treatment; lastly, I have never seen anything but harm follow a series of severe reactions.

PROGNOSIS.

If the neoplasm is visible or palpable, increase or decrease may be obvious. Increase in size, at first, may not mean that the neoplasm is growing, it may be due simply to increase of junction tissue. Gain in weight, when weight has been lost, is favourable. Loss of pain is good, especially if it continues, but most cases get some relief of pain even though ultimately not responsive. The general condition of the patient as regards appetite and gain in strength is important; these may be unsatisfactory in the specially sensitive neoplasms when doing well. Sometimes, when breakdown is rapid, the patient appears to be going downhill rapidly; but if the masses are found to be diminishing, treatment should continue, though ionization should either be given longer intervals or be stopped for a time. At times the patient is nearly killed from the absorption of the products of breakdown. The following case is an example:—

Par.—Sent by Mr. Hey Groves. A large mass about the size of a melon but flatter, was situated on the ilium, extending up into the lumbar muscles and down into the thigh, growth rapid. Biopsy diagnosed a highly malignant endothelioma (Professor M. Stewart) or polymorph sarcoma (Dr. A. L. Taylor). Reaction to small doses of SSe was marked, and consistently smaller doses were given; reaction to X rays was also great and the patient was reduced to 1 H. unit. The mass rapidly began to diminish, but general toxæmia became so marked that ionization was stopped at 6 H. units. He was then very ill, had no strength, and was unable to retain food. For two months small doses of D4S were given weekly, as reaction from this is less and slower than from the newer colloids. He gradually picked up and was put on alternation, with gradual increase of dosage as his tolerance

permitted. The mass disappeared and his health became normal except for a gradual increase of adiposity—this happens not infrequently. He has had no treatment for eighteen months and is back at work.

The reappearance of chronic rheumatism in a patient appears to be of very favourable prognosis. But before diagnosing rheumatism, it is necessary to make sure that the suspected rheumatism is not due to bony or neural metastasis. A rheumatic subject loses his rheumatism as cancer develops, and may experience it again after removal of the growth by whatsoever method. This regression of rheumatism appears to be of value in dating the approximate onset of the neoplasm; but it is not absolute, as I have seen rheumatism return shortly after operation, in a case which recurred within six months. I have observed this alternation of rheumatism and cancer for about four years; up to the present I have had no cause to alter my opinion.

For those who wish to have more objective guides to prognosis certain laboratory tests are available; they are not necessary, but we apply them as routine. These tests are:—

1. *Blood Lipase, Rona Technique*.¹—This lipase, not the so-called lipase of Shaw Mackenzie, tends to be high in cancer, but not invariably. We have used this test in every case since 1927 and find that a lowering level is usually favourable, an increasing level usually a sign of advance in growth.

2. *Blood Cytology*.—An increase, absolute, not relative, of small lymphocytes and eosinophil cells is also favourable. It is regarded as evidence of reactivity, which is favourable, but it does not imply that the reactivity will be adequate to cause complete inhibition of the growth. On the other hand, absence of this finding practically always means that no response to treatment is taking place.

3. *Blister Test*.—A standard size cantharides blister, about one inch (but provided the size is always the same it does not matter), is applied between elbow and shoulder the night before inspection. The next day the fluid is removed if a take has occurred, the amount is roughly estimated, and a cell-count is made. This is regarded as a test of the reactivity of the patient's tissues to a standard irritation; the finding of a good take, with increase of endothelial, lymphocyte, and eosinophil cells, is also favourable with the limitations given under blood cytology. Increase of epithelial cells is not of any importance.

Duration of Treatment in Unfavourable Cases.—The duration of treatment for cases responding favourably has already been given. In a case apparently not responding I go on until the patient is definitely moribund, even though all the prognostics are bad, for occasionally a patient will begin to improve in the end. Example:—

Ken.—Carcinoma of rectum, sent by Mr. A. Jackman. Previous excision with radium application afterwards. Recurred, and pelvis reported full of neoplasm. For six months this man appeared to be steadily going downhill and all the tests were adverse; lymphocytes, etc., did not rise, blister result was bad, blood lipase increased from 13 to 21 per cent, and he appeared likely to die soon. Yet, on continuing, he began slowly to improve, and he was discharged nearly two years ago; he is back at work as a bricklayer.

THE RESULTS OF TREATMENT.

The results are dated from May, 1931, at which time the treatment as described was started. SSe had been given before this date, but R.A.S. and alternation only started then. The first series is from May, 1931, to September, 1932. The second series started 1932 and continues to the present time. Series 1 consists of 93 cases. No selection of any sort was applied to this series—every patient was accepted if there was room. In Series 2, as has been noted, the patient has to undertake to attend weekly for a year or more, and those of feeble mentality, not judged able to co-operate, are excluded. Series 1 (*see Table I*) includes 7 cases which had been treated on the earlier system, but, being not satisfactory, were given the present treatment; this

Table I.—RESULTS IN SERIES 1.

NAME INDEX	DURATION OF TREATMENT	TYPE OF GROWTH	TOILET
Failures (29 cases).—			
Arn. ..	12	Mammary	Yes
All. ..	24	Mammary	Yes
Bes. ..	10	Ovarian	Yes
Bon. ..	19	Rectum	Colostomy
Bri. ..	4	Œsophagus	No
Bra. ..	16	Rectum	Colostomy
Car. ..	4	Bronchial	No
Cai. ..	4	Rectum	Colostomy
Edm. ..	5	Polymorph-cell sarcoma	No
Gay. ..	5	Lymphosarcoma	No
God. ..	7	Carcinoma of tonsil	No
Hal. ..	12	Osteosarcoma	No
Hob. ..	7	Mammary	No
Hil. ..	8	Prostate	No
Han. ..	5	Penis	No
Ile. ..	11	Seminoma	No
Jew. ..	5	Rectum	Colostomy
Jon. ..	7	Mammary	?
Ket. ..	3	Bronchial	No
Mil. ..	5	Sarcoma of pelvis	No
Mon. ..	6	Sarcoma, retroperitoneal	No
Phc. ..	11	Mammary	Yes
Pea. ..	9	Rectum	Colostomy
Tom. ..	30	Mammary	No
Tur. ..	9	Rectum	Colostomy
Tra. ..	6	Ovarian	No
Tur. ..	4	Rectum	Colostomy
Wat. ..	3	Labia majora	No
Tur. ..	4	Rectum	Colostomy
Still on Treatment (5 cases).—			
Har. ..	33	Rectum	Stationary
Hal. ..	30	Mammary	Very good
Han. ..	16	Myeloid leukaemia	Doubtful
Par. ..	19	Carcinoma of tongue	Bad
Sma. ..	48	Mammary	Good

Dropped Out (18 cases).—Very similar in types to those above. Five were doing well.

Inadequate Treatment (25 cases).—Types similar except one gastric carcinoma. Average duration of treatment 6 weeks.

NAME INDEX	DURATION OF APPARENT CURE	TYPE OF GROWTH	CAUSE OF DEATH
Deaths from Other Causes (3 cases).—			
Bar. ..	Months 5	Luteoma	Accident
Car. ..	18	Mammary	Influenza
Hew. ..	12	Mammary	Influenza
Cases of Apparent Cure (15 cases).—			
Bat. ..	18	Rodent	No
Dav. ..	15	Sarcoma of pubis	No
Eva. ..	15	Ovary	No
Gay. ..	20	Mammary	Yes
Hob. ..	19	Mammary	Yes
Ken. ..	17	Rectum	Colostomy
Mai. ..	15	Sarcoma of orbit	Enucleation of eye
Mos. ..	8	Ovarian	No
Par. ..	19	Sarcoma of ileum	No
Pie. ..	20	Ovarian	Yes
Phi. ..	7	Mammary	Yes
Phe. ..	7	Mammary	Yes
Ped. ..	7	Melanoma of eye	Enucleation
Sav. ..	18	Mammary	Yes
She. ..	6	Rodent of orbit	Enucleation

is why some of the durations of apparent cure almost approach the duration of the present method. Of these 93 cases, 18 dropped out, for the reasons noted under the present selection—i.e., stupidity, or distance, or expense; 5 of these cases were doing well and some of them stopped because they thought they were cured. Then 5 other cases are still receiving treatment; of these, 2 are doing well and are likely to be discharged later. Excluding these cases there are left 70 to which treatment has been given.

Of this series of 70 cases, 23 had quite inadequate treatment, the average duration being only six weeks; that is, they were extremely advanced cases which would not have been accepted if any selection had been made. If they are excluded, there remain 47 cases in which treatment has been adequate; in 29 cases treatment has failed, in the end, and all are dead. This leaves 18 cases, of which 15 are discharged as apparent cures. Then 3 cases, apparent cures, have died from influenza in two instances and street accident in one, in which autopsy disclosed no growth.

That is, of the whole series, apparent cure has resulted in 20 per cent. Excluding the cases still on treatment and those which gave up, the figure rises to 26 per cent. If only the cases which received adequate treatment are counted, the figure rises to 38 per cent.

Series 2, or those treated since September, 1932, is more difficult to assess; there are 62 cases (*see Table II*). Selection has been made, but only as regards mentality and ability to attend. Of these cases 12 have started treatment too recently for any decision to be made. Then, in spite of the selection, 7 cases dropped out; one was doing reasonably well. Inadequately treated cases number 15. average duration of treatment being four weeks. This leaves 28 cases to which adequate treatment has been given. Of these.

12 are dead, 4 are still receiving treatment, but are doing badly ; 9 are doing well, amongst them being several who are being observed prior to discharge ; 3 cases have been discharged as apparent cures. That is, the results are at least as good as in the first series.

Table II.—RESULTS IN SERIES 2.

NAME INDEX	DURATION OF TREATMENT	TYPE OF GROWTH	TOILET
------------	-----------------------	----------------	--------

Recently started on Treatment (12 cases).

Stopped Treatment against Advice (7 cases ; 1 doing well).

Received Inadequate Treatment, Average Duration, Four Weeks (15 cases).

Received Adequate Treatment : but Dead (12 cases).—

	Months		
Bon. ..	9	Mammary	Yes.
Bri. ..	3	Carcinoma of uterus	No
Cra. ..	3	Carcinoma of tongue	No
Hol. ..	10	Mammary	Yes
Jon. ..	9	Mammary	?
Mea. ..	10	Ovarian	No
Poo. ..	7	Buccal carcinoma	No
Par. ..	4	Ovarian	No
Sen. ..	7	Mammary	Yes
Swi. ..	24	Mammary, bilateral	Yes
Sic. ..	7	Branchial	No
Tud. ..	10	Ovarian	No

Received Adequate Treatment, still Alive, but Doing Badly (4 cases).—

Edw. ..	6	Primary, hepatic	No
Ham. ..	5	Oesophagus	No
Sam. ..	4	Oesophagus	No
Web. ..	5	Tonsil	No

Received Adequate Treatment, Doing Well (9 cases).—

Bry. ..	16	Melanoma of eye	Enucleation
Bur. ..	5	Osteosarcoma	No
Bis. ..	4	Mammary	Yes
Fow. ..	8	Mammary	No
Poo. ..	14	Mammary	Yes
Sea. ..	5	Multiple myeloma	No
Tar. ..	11	Mammary	No
Tan. ..	8	Mammary	Yes
Wes. ..	13	Ovarian	Yes

Discharged, Apparent Cure (3 cases).—

	DURATION OF APPARENT CURE		
	Months		
Hem. ..	3	Mammary	Yes
Sti. ..	11	Osteosarcoma	Yes
Wil. ..	3	Mammary	Yes

These bald figures do not give quite a true picture of the results, for many of the cases recorded as failures showed some good result. For instance, amongst those who dropped out was this case :—

Hor.—This patient was seen by many, including the radium commissioners, for she was treated in the radium ward, owing to scarcity of beds. She showed on

admission a fungating septic breast with large glands in the axilla and over the clavicles. Her liver was an enormous craggy mass. Her lungs showed large masses in both sides and she had a spontaneous fracture of one femur. She was in early cachexia. 'Toilet' was done to breast, and she was then treated as I have described. She steadily improved, the fracture healed with 3 in. of shortening, the liver masses disappeared, the lung masses became no longer visible, and the only sign of disease left was a right supraclavicular mass, which appeared to be calcified. She got up, and went home, being able to walk with a limp. She attended for a time and was seen by Mr. Hey Groves, who advised surgical boots. She received from him the impression that she was quite cured, and nothing would persuade her to attend for further treatment. She was rather poor and had to attend in a hired car from her home thirty miles away. She did well for about six months, then relapsed and died.

Another case recorded as a failure was the following :—

Swi.—On admission this patient had a foul fungation of the right breast and a large central carcinoma of the left; there were many axillary and supraclavicular glands. She was much wasted and very anæmic from sepsis and hæmorrhage; she had been bedridden for some time. Mr. H. Chitty did a 'toilet' to both sides, and then she was put on the D4S system, as she lived in the Midlands. She did well for six months and went home, living fairly comfortably and able to do some work, quite as much as her advanced age allowed. Eight months later a small recurrence appeared about the scar, so she came to Bristol and was started on ionization, of which she had a full course; this appeared to arrest growth and she returned home and again received D4S. She was well for about six months, when again recurrence appeared. She returned to Bristol and had another course of ionization, after which she was again comfortable. The later history is that recurrences re-appeared and she would not again attend for treatment. A failure, but a dying woman was given about eighteen months of relatively happy and useful life.

Many similar cases could be recorded of temporary improvement amongst the failures. I will not labour the factor of relief from pain, but it is pronounced in many of the patients.

This is the new method, described as fully as possible. The work is done in public and has been inspected by a number of distinguished surgeons. The method has produced these results. If the same results are not obtained at other clinics, then let those clinics look to their technique and their ability to drive a difficult treatment through to results as good as, or, I hope, better than, these.

Lastly, and with great appreciation, I have to thank the other members of the team and the radiological staff for their help. Some have contributed more than others, but all have done their best; for that reason I think it would be invidious to single out workers by name, with the exception of Mr. E. Lloyd, M.P.S., to whose ingenuity and knowledge of chemistry the perfection of these preparations is due. The cost of this work has been borne by the Bristol Royal Infirmary Cancer Research Fund; a small grant has recently been made by the Imperial Cancer Research Fund.

REFERENCES.

- ¹ *Chemotherapeutic Researches on Cancer.* London and Bristol: J. W. Arrowsmith Ltd.
- ² *Brit. Med. Jour.*, 1929, ii, 799.
- ³ *Lancet*, 1930, i, 906.
- ⁴ *Ibid.*, ii, 389.

**BILHARZIA DISEASE IN ENGLAND:
THE CYSTOSCOPIC APPEARANCE OF THE BILHARZIA BLADDER
BEFORE AND AFTER INTRAVENOUS INJECTIONS
OF SODIUM ANTIMONY TARTRATE.**

By J. B. CHRISTOPHERSON,

PHYSICIAN (TROPICAL DISEASES), ROYAL MASONIC HOSPITAL, LONDON.

AND R. OGIER WARD,

ASSISTANT SURGEON, ST. PETER'S HOSPITAL FOR STONE, LONDON,

THE case now under review was a young man, M. S., aged 20. He had landed perfectly fit in Cape Town in February, 1929. Thence he proceeded to Mapunga, seventy miles north of Salisbury in Southern Rhodesia, to work on a tobacco estate, and whilst so occupied he had bathed frequently in a tributary of the Mazoe River—an auxiliary of the Zambezi.

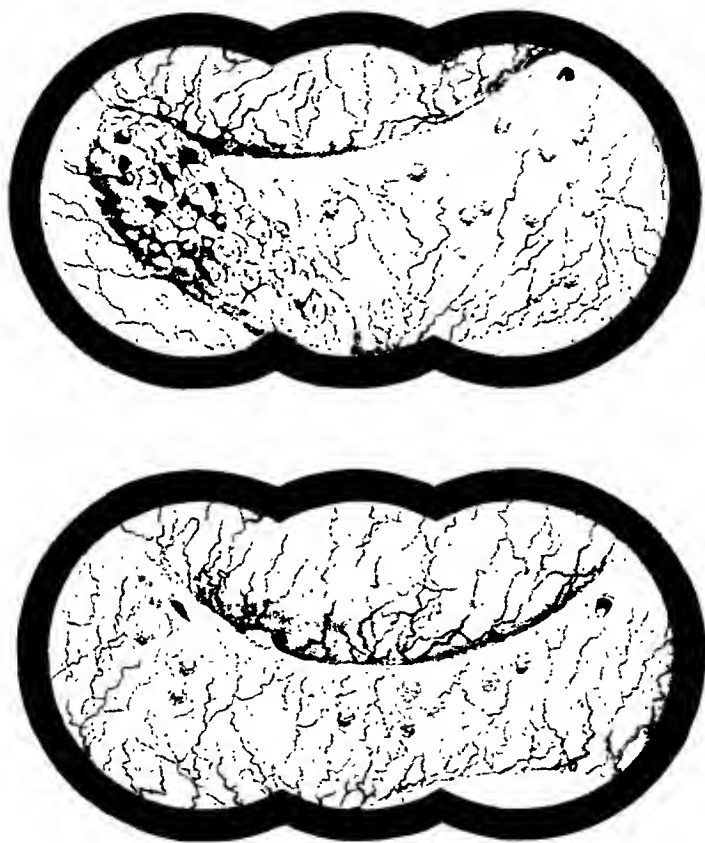
He appears to have contracted dysentery and bilharzia disease in June, 1929, and was admitted to the local hospital. During his illness his temperature rose to 104° , he passed several small clots of blood in his urine, and once had retention of urine for thirty-six hours. Quite unaware that he was suffering from bilharzia infection he returned home to England in November, 1929.

During the period June, 1929, to October, 1930, several attacks of hæmaturia occurred, some of which were considerable and were accompanied by the passage of clots, though all were of short duration. He also suffered from attacks of pain in both loins, one of which immediately followed a severe jolting on a tractor. On the whole the pain was worse in the left posterior renal angle than on the right side. In February, 1930, a cystoscopy and a left pyelogram were carried out, and the patient was told that they revealed nothing abnormal.

He was first seen by one of us (R. O. W.) on Oct. 10, 1930, and ova of *Schistosoma hæmatobium* were discovered in the urine on Oct. 16 by Dr. Cuthbert Dukes. The urine was slightly turbid, acid, specific gravity 1024, and contained terminal spined ova, though not in great numbers; these were found to be viable. Pus and blood were also present; no organisms were seen in films, but some streptococci and staphylococci were cultured. The results of blood examination are discussed later. A cystoscopic examination was made at this time, and Fig. 394 shows the local condition which was disclosed.

The bladder was almost normal except for the changes depicted, though a slight degree of congestion of the mucosa was present. The outstanding feature was the intense bullous œdema which surrounded the right ureteric orifice and extended on to the trigone below it. Amongst this œdema and

elsewhere on the trigone were vesicles, quite distinct from those bullæ, which are characteristic of a simple œdema in that they were of a peculiar yellow colour. Their presence is diagnostic of vesical bilharzia. Around the ureteric orifice were several small hæmorrhagic extravasations of recent origin; the opening of the ureter itself could just be recognized. The left ureter was not quite normal, being somewhat rigid and appearing to be in a state of chronic inflammation.



FIGS. 394, 395.—Bilharzia disease of the bladder before treatment, and one month after treatment with sodium antimony tartrate. The yellow nodules in Fig. 395 are the dead ova working their way through into the bladder cavity. They do not indicate active bilharzia disease.

A course of intravenous injections of sodium antimony tartrate was begun on Oct. 23, 1930, starting with $\frac{1}{2}$ gr. and 1 gr. on the following day. The following injections were given on alternate days, the dose increasing $\frac{1}{2}$ gr. each injection till 2 gr. was reached, then seven doses of 2 gr. each were given, and finally four doses of $2\frac{1}{2}$ gr.—a total of 28 gr. in twenty-eight days (four injections were made each week, excepting one week, when three were given); the patient remained in bed on injection days. The complications

which occurred during the injections were the customary painful dry cough accompanied by pain in the chest and a feeling of discomfort in the throat immediately after the antimony injection. After a few days, in order to prevent this, the patient was given $\frac{1}{4}$ gr. of omnopon (intramuscularly) ten minutes before the antimony injection. This was successful in abolishing the cough, the pain in the chest, and the discomfort in the throat—formidable inconveniences of antimony tartrate injections. He complained of a feeling of sickness in the evening once after the injection, but beyond those mentioned there was no complication during treatment. He escaped the muscular pains, often paralysing in their severity, which frequently accompany antimony tartrate injections and are particularly liable to follow the larger doses. Possibly this was due to the administration of omnopon. As usual after a total of 9 gr. sodium antimony tartrate had been administered, ova and blood disappeared from the urine, and neither have reappeared in the specimens subsequently investigated at intervals over a period of three years.

The pulse-rate was taken before and five minutes after each injection, and the blood-pressure was also registered before and after the injections. This is not usually done, but investigations of pulse and blood-pressure during the course of injections yield valuable information on the depressing effects of antimony tartrate injections and constitute a useful control.

The second cystoscopic examination was made on Jan. 23, 1931, nominally one month after the last injection (*Fig. 395*). The bilharzia tissue had disappeared and the mucous membrane of the bladder appeared to be quite normal except for the small circular yellow vesicles scattered here and there like amber beads.

The patient was kept under observation after treatment and was last seen as recently as June, 1933, three years after the course of antimony tartrate, when he was working on his own farm in Northumberland, in normal good health, free from all urinary symptoms since the course of injections.

DISCUSSION.

A course of approximately 30 gr. of antimony tartrate, given intravenously in the dose and at intervals originally recommended by one of us (J. B. C.¹) in 1918, is the generally accepted treatment of choice for human bilharzia disease in all its forms; the sodium salt is usually preferred to the potassium. The course of four or five weeks is somewhat long, but, provided the full course of 28 to 30 gr. (adult) can be given within that time, the cure is almost unfailing.

Antimony tartrate administered for bilharzia should be given in increasing daily doses for the first four days until 2 gr. per dose has been reached, then on alternate days 2 or $2\frac{1}{2}$ gr., the aim being to reach as rapidly as possible the concentration of antimony in the blood necessary for the parasitidal effect. Given timidly in too small doses, or given at too long intervals, antimony fails to produce the lethal effect on the parasites, the more resistant of which have a tendency to become antimony-fast.

A young man recently seen by one of us in London had contracted schistosomiasis (*S. japonicum*) near Shanghai whilst bathing in a tributary of

the Yangtze-kiang River. He was admitted into the local hospital and given a course of emetine. This was followed by a course of neo-stibosan (a pentavalent synthetic antimony derivative). When these failed there followed three separate courses of fouadin, the first of which was a modified course. Fouadin is neo-antimosan (Bayer), a trivalent synthetic antimony derivative. This not having the desired effect, a course of sodium antimony tartrate or tartar emetic was given. When he landed in England his faeces still contained scanty living ova, no doubt owing to the fact that some of the worms had become antimony-fast, the most powerful *bilharziacide* not having been used early enough nor according to the generally accepted technique.

Organic Preparations of Antimony and Bilharzia Disease.—Chemists searching for a compound producing the maximum parasito-tropic and the minimum organo-tropic effects in bilharzia have introduced neo-stibosan—an amino compound of antimony—a comparatively non-toxic pentavalent substance—effective in the protozoal infection leishmaniasis. When tried for bilharziasis neo-stibosan failed and was abandoned. Pentavalent organic preparations of antimony are not sufficiently parasito-tropic for the metazoal parasite bilharzia.

The trivalent Bayer preparation neo-antimosan (sodium antimony pyrocatechin di-sulphonate) named fouadin—after King Fouad of Egypt—has been tried since 1928. It is recommended by medical authorities in Egypt, but it has not altogether justified the praise of its Egyptian sponsors. Recent letters from South Africa report hepatic complications and loss of weight following the injections of fouadin for bilharzia. Fouadin is given by intramuscular injections and it is a useful drug for children whose veins are small, and for adults when antimony tartrate is contra-indicated.

Sodium and potassium antimony tartrate are in my opinion (J. B. C.) the parasitocides of choice for all cases of bilharziasis when it is possible to use them and when not contra-indicated.

The blood-picture in the present case was that of anaemia associated with eosinophilia: Haemoglobin 72 per cent, colour index 0.9, eosinophils (before injections) 7 per cent. Two days after the completion of the injections the eosinophils were 12 per cent. Two months later eosinophils were 2 per cent, and five months after the completion of the course the differential count was normal: Polymorphonuclears 63 per cent, lymphocytes 31 per cent, large mononuclears 2 per cent, eosinophils 3 per cent, basophils 1 per cent.

The differential blood-count in bilharzia is of very definite clinical value—eosinophils may reach 70 per cent. Bilharzia and trichinosis amongst tropical diseases give the highest eosinophilia. Eosinophilia, however, has no relation to the number of parasites present—it registers the effort of the organism to combat the parasite. Eosinophilia increases as injections proceed. Eosinophilia three months after the termination of antimony injections argues the patient un cured.

In *Fig. 395* the small round yellow vesicles like small canary seeds or the halves of small amber beads stand out on the clean mucosa with no surrounding inflammation. In *Fig. 394* these nodules have the same general characters, but some of them are accompanied by slight inflammation. We do not consider them to indicate activity due to living ova: on the contrary

we believe that they are caused by the existence of ova which are dead, and which will ultimately be shed into the bladder cavity, for they are seen to be present after complete and successful treatment. We think this observation worthy of record.

We have already called attention to the clinical importance of the differential blood-count in cases of bilharzia disease.

There is illustrated in this case a third point—small, though one of considerable therapeutic importance in the clinical treatment of this disease. The distressing useless painful cough, and discomfort in the throat and chest, experienced immediately after individual injections may be alleviated or abolished by $\frac{1}{4}$ gr. of omnopon given ten minutes before injections. Possibly also the painful, alarming, paralysing muscular symptoms which occur later in the course may be eliminated by the same means.

Bilharzia disease—schistosomiasis—is not endemic in this country. There is on record no well-established case of the disease having been contracted in the British Isles. Bilharzia, however, not infrequently crops up in England, but is always imported. Biologically it would not appear impossible for the disease to thrive and to pass from one person to another in England. There are stretches of fresh water, e.g., at Frensham and Fleet in Surrey, and in other localities, where soldiers bathe whilst at home from abroad in water which harbours species of snails, which, given the opportunity, might function as the necessary intermediate hosts; but there are few days in the year when the miracidium in the ovum would hatch out and survive in the free state and infect the snails. The British climate preserves the country from endemic bilharzia disease. The cases which are seen in England come from the Far East—China, Japan, Formosa (*Schistosoma japonicum*), from Egypt or one of the many infected areas in Africa, from Palestine, Mesopotamia (*Schist. hæmatobium* and *mansoni*), or the West Indies (*Schist. mansoni*), and an occasional case (*Schist. mansoni*) from South America. Owing to increasing facilities for travel and trade, bilharzia disease is not an uncommon visitor to this country, though usually it does not occur in a serious form.

REFERENCES.

- ¹ CHRISTOPHERSON, J. B., "The Successful Use of Antimony in Bilharziasis", *Lancet* 1918, Sept. 7; "Intravenous Injections of Antimonium Tartratum in Bilharziasis", *Brit. Med. Jour.*, 1918, Dec. 14; "Laboratory and Other Notes on Seventy Cases of Bilharzia Treated at the Khartoum Civil Hospital by Intravenous Injections of Antimony Tartrate" (with J. R. NEWLOVE), *Jour. Trop. Med. and Hyg.*, 1919, July 15.

SPINDLE-CELLED MESENTERIC TUMOURS: WITH REMARKS ON SIMILAR RETROPERITONEAL TUMOURS.

BY H. A. PHILLIPS, MELBOURNE.

Definition.—Modifying Twistington Higgins and Lloyd,¹ who wrote on mesenteric cysts, it would seem advisable to define a 'mesenteric' tumour as one which occurs as a primary growth in or near the mesentery and which has not originated in a normal retroperitoneal organ or gland. It is proposed to call these 'paramesenteric tumours', and such tumours will therefore embrace: (1) Those of the retroperitoneal tissues; (2) Those growing from the connective tissues between the leaves of any of the peritoneal folds, omenta and mesenteries.* Confusingly enough, the cyst described by the above authors as mesenteric was in point of fact, retroperitoneal. The reason for the necessity of a term which will include both retroperitoneal and mesenteric tumours and cysts is obvious: (1) These structures have a common anatomical relationship to the peritoneum—they are all subperitoneal: (2) The same theories of developmental origin are applicable to them; (3) The actual pathology of solid tumours, as will be shown below, is similar.

Historical.—It appears that whilst Benevieni was the first to report a mesenteric cyst (sixteenth century), Morgagni (1761) was the first to note a solid mesenteric tumour, although this distinction is often given to Portal (1803). Hertzog² in 1897 collected from the literature the first series of reported cases (57). Vance³ in 1906 added a further list of 27 cases. In the British literature, Greer⁴ in 1911 records a series of 33 cases of fibromatous tumours culled from various sources. In the German, Szenes⁵ has analysed a series of 60 cases (1918). The most recent account is given by Rankin and Major.⁶

Now there has been no little confusion and much repetition in this literature of solid tumours. In some cases lymphomatous and carcinomatous tumours have not been excluded; cases have been duplicated into several series, and the histology and pathological genesis of the tumours have received scanty notice, American authors in particular contenting themselves with a discussion of the clinical features. Probably the best modern series is that of Rankin and Major.⁶ It comprises 22 mesenteric tumours of the Mayo Clinic, 7 of these being cysts and 15 solid tumours. No tumour of lymphatic-gland origin entered into the series. This excellent review, though, like many of its predecessors, gives no after-histories of its cases and is content to follow the older terminology.

* The extra-ligamentous broad ligament tumours are tacitly excluded, as their pathology is so closely bound up with that of the uterus and ovary. No doubt, however, an analogy exists between the fibromata and myomata of this region and the paramesenteric tumours here discussed.

Incidence.—To show how rare these tumours are, and to compare their incidence with that of other paramesenteric lesions, the following figures are given. Of the last 4500 autopsies performed at the Alfred Hospital, Melbourne (since 1914), there were :—

- 1 case of tumour of the gastro-hepatic ligament.*
- 3 cases of retroperitoneal sarcoma.*
- 1 case of adrenal neuroblastoma.
- 3 cases of retroperitoneal hydatid.
- 6 cases of Hodgkin's disease showing large retroperitoneal glandular enlargements as a prominent feature.
- 4 cases of lymphosarcoma of the aortic and lumbar glands.
- 2 cases of retroperitoneal hæmatoma due to ruptured aortic aneurysm.

Unfortunately a similar résumé of the in-patients who did not come to post-mortem in this thirty-year span cannot be included. But of the 19,788 in-patients admitted from 1930-3 there were :—

- 4 cases of retroperitoneal tumour—proved by operation.*
- 1 case of tumour of the great omentum—proved by operation.*

Curiously enough there is no record of a single case of mesenteric or retroperitoneal cyst.

The Museum of the Royal College of Surgeons has only two specimens of mesenteric tumour, the one a fibroma and the other a lipoma (Nos. 6151-1 and 6155-2).

There is no mention in the literature of any percentage of incidence of mesenteric or retroperitoneal tumour.

Résumés of the clinical features of the cases noted above, followed in each instance by the descriptive pathology and after-history, are given below. It will be shown that, despite their variation in site, these tumours have a common pathology.

‘ MESENTERIC ’ TUMOURS.

Case 1.—Tumour of gastro-hepatic ligament.

C. J., female, aged 63, and multiparous. She had noticed a swelling in the abdomen for four years. In the last three months it had caused her a little discomfort and she was definitely more dyspnoic and pale than previously and had grown weaker.

ON EXAMINATION.—This tumour was found to be a hard, spherical, immobile mass dull to percussion and without fluid thrill. The flanks were resonant. The tumour filled the abdomen and was palpable per vaginam, where a small atrophic uterus was pushed back by it. A diagnosis of ovarian cyst was made with some misgivings, and a laparotomy was performed by the honorary gynaecologist.

OPERATION.—About a pint of free fluid escaped on opening the abdomen, and the tumour was found to be cystic in one place; after the aspiration of a pint of brownish fluid the tumour could be everted, when it became evident that there

* Seven of the nine cases marked above with an asterisk form the basis of this paper. Two cases of retroperitoneal sarcomata were not included, because there was no microscopic section for one, and the other was microscopically a round-celled tumour and regarded as possibly of lymphatic-gland origin. It is very important in these cases, if possible, to identify the lymph-glands as normal.

was no ovarian pedicle. It was necessary to strip the upper pole from the posterior wall of the stomach, during which the latter was inadvertently opened, there being no covering peritoneum visible. The tear was quickly repaired and a pedicle defined in the depths of the abdomen. This was hastily ligated, the tumour removed, and the abdomen closed as the patient's condition had become rather alarming. She died twenty-four hours later from post-operative shock.

AUTOPSY.—At post-mortem the uterus and adnexa were normal, and it was definitely established that the tumour had developed in the gastro-hepatic ligament and that the pedicle contained hypertrophied branches of the gastroduodenal and right gastric arteries. There was no other tumour in any other part of the abdomen or in the thorax or brain. The tumour originating in the gastric border of the gastro-hepatic ligament extended in such a way as to obliterate the lesser omental sac, pushing the stomach forwards and upwards and the transverse colon posteriorly, remaining 'peritonealized', however, on all surfaces except that in contact with the stomach, and being also enclosed by the outer two layers of the attenuated great omentum, somewhat distorted by adhesions.*

PATHOLOGY.—Macroscopically the tumour was ovoid, 30 cm. (12 in.) in length and 17.5 cm. (7 in.) in breadth and depth. It weighed 4196 grm. (9 lb. 4 oz.). Its surface was greyish-white and was covered with peritoneum except at the region of the pedicle, where it was irregular and particularly cystic (*Fig. 396*).

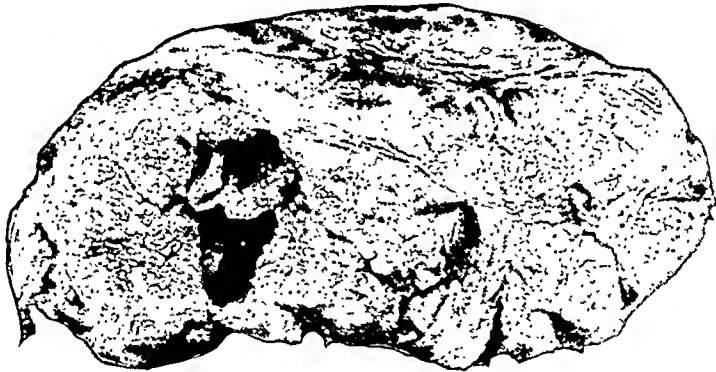


Fig. 396.—*Case 1.* Macroscopic appearance of tumour.

In cross-section most of the central portion of the tumour consisted of soft yellowish caseous material into which passed bands of firmer fibrous tissue. There was a cortex of about 5 cm. in width which in some parts could be described as fleshy, but in others appeared more like fibrous tissue. A few small cysts were visible in the fibrous areas.

Microscopically the bulk of the tumour was composed of structureless caseous material taking up the counterstain. Many areas of the tumour consisted of bundles of spindle cells with very few intervening fibres of connective tissue demonstrable by van Gieson's staining (*Fig. 397*). Many of these cells possess nuclei which stand out as long rods with blunt ends (*Fig. 398*), resembling therein unstriated muscle fibres of embryonic type. They stain brown with van Gieson's. Other areas consist mostly of myxomatous tissue with a few of these spindle cells interspersed amongst it. There are visible several dilated spaces lined with endothelium and resembling lymphangiomatous tissue. The small cysts visible macroscopically had no lining membrane. They are therefore probably degenerative.

* McW. Millar⁷ has reported a similarly situated tumour which he terms a "pedunculated extra-gastric leiomyoma of stomach with hæmorrhagic degeneration". It would appear to resemble in pathology the spindle-celled tumours described in this paper.

The biopsy section of the pedicle tissue still remaining in the gastro-hepatic ligament was found to show similar microscopic findings. The tumour was considered to be a fibromyxomyoma or mixed tumour of embryonal type.



FIG. 397.—Case 1. Microscopic appearance of tumour showing spindle cells.



FIG. 398.—Case 1. Section showing nuclei.

Case 2.—Tumour of great omentum.

H. P., female, aged 73. She had noticed a lump in the right iliac fossa for four months. It became larger, till at the time of examination it filled the abdomen. She complained of slight loss of weight and mild indigestion, but nothing else of importance.

ON EXAMINATION.—There was a large, spherical, hard tumour the size of a human head filling up the lower abdomen. The flanks were resonant, and pelvic examination revealed nothing untoward. The tumour was immobile. Examination of the blood indicated a secondary anæmia, there being 65 per cent of hæmoglobin, 4,130,000 erythrocytes per cmm., and considerable anisocytosis in the stained film.

OPERATION.—At operation the tumour was found to be in the layers of the great omentum but very adherent to surrounding structures. It was considered unwise to remove it, so a portion was excised for section and the abdomen closed.

The patient had an uninterrupted convalescence and was discharged fifteen days later. She died eight weeks later, no post-mortem examination having been made.

HISTOLOGY.—Histologically the greater part of the section consisted of myxomatous tissue in which there were numerous areas showing various degrees of necrosis and hæmorrhage. There was at the periphery of the tumour a cortex 3 or 4 mm. wide of much more cellular tissue consisting of irregularly arranged spindle and oval cells in a fibrillar matrix. There were no muscle fibres though the cells stained brown with van Gieson's, and no blood spaces of sarcoma type.

It appears probable that in the more central portions the tumour was undergoing an ischæmic form of myxomatous degeneration even to the point of caseation. The microscopic appearances, taken into consideration with the short clinical history, would suggest a diagnosis of degenerating fibroma.

RETROPERITONEAL TUMOURS.

The following are the case histories and pathology of four similar spindle-celled retroperitoneal tumours.

Case 3.—Pedunculated tumour of retroperitoneal pouch.

P. R., male, aged 50. For ten years this man had noticed a tumour in his lower abdomen. It had occasioned him no discomfort, and over most of that period

did not seem to be enlarging. However, for the last two years he had been troubled by a frequency of micturition, sometimes two-hourly, through the day and night. There were no symptoms referable to the bowel and the patient had not lost weight.

ON EXAMINATION.—There was an easily palpable oval mass lying across the lower abdomen and occupying both iliac fossæ. A distended bladder could be made out anterior to the tumour, and the presence of small bowel also anterior was suggested by a resonant percussion note over the upper half of the mass. Per rectum the tumour appeared to be lying in the pelvis, pressing against the outer wall of the rectum, though not attached to it. Bimanually it was freely mobile and non-fluctuant, and its surface felt hard and irregular.

OPERATION.—At operation it was discovered to be a retroperitoneal solid tumour growing between the bladder and rectum and bulging forwards to become pedunculated. The peritoneum and pelvic fascia over the narrowest part of the



FIG. 399.—Case 3. Macroscopic appearance of tumour.

tumour was incised and the tumour shelled out. There was slight hæmorrhage from the cavity remaining, but this was easily controlled by pressure. The abdomen was drained for twenty-four hours.

The patient made an uneventful recovery, and all his symptoms, including the frequency of micturition, entirely disappeared.

Two years later the abdomen was opened again for the relief of intestinal obstruction due to adhesions, and no recurrence was discovered. The patient was alive and well one year after this second operation.

PATHOLOGY.—The tumour when removed weighed 1730 grm. (3 lb. 11 oz.). It was 25 cm. (10 in.) in length, and 15 cm. (6 in.) in breadth and depth. It was clothed in peritoneum except at the base (*Fig. 399*), where there were some irregular cystic projections.

On section it was solid and greyish-white in appearance. It possessed a thick capsule and was intersected by numerous trabeculæ of fibrous tissue enclosing gelatinous areas into some of which hæmorrhage had occurred.

Microscopically the tumour was also very similar to that of *Cases 1 and 2*. It consisted of a myxomatous tissue with varying admixtures of spindle cells. The cysts are degenerative in type, and have no lining membrane. There were no fully formed blood-vessels to be seen, but collections of erythrocytes were occasionally present in spaces lined by a single layer of endothelial cells. The inadequacy and immaturity of the blood-vessels is a common feature of this type of tumour, and explains the ease with which degeneration and caseation occurs.

Case 4.—Retroperitoneal sarcoma of left lumbar region.

H. W., male, aged 42. Twelve months previously his left leg had started to swell; at first only in the evenings, but later it became continuous and extended up the leg. For one month he had noticed his abdomen swelling. There was no alteration in his bowel or micturition history, but there had been some loss of weight.

ON EXAMINATION.—The abdomen was distended. The swelling was due to the presence of free fluid, distended bowel, and of a large mass mostly on the left side. Both flanks were dull, but there was a band of resonance immediately to the right of the tumour.

There was a moderate degree of anæmia. Numerous investigations were carried out and paracentesis was performed, a gallon of fluid being removed. Eventually a laparotomy was performed.

OPERATION.—At the operation much free fluid escaped and the tumour was found to be a retroperitoneal growth. When needed a large quantity was removed. When incised the tumour was very hæmorrhagic; the bleeding area demanded packing. The tumour was too extensive for removal.

About three weeks later the wound broke down and a fæcal fistula developed. The patient gradually went downhill, and died three months later.

AUTOPSY.—At the autopsy the whole of the small intestine was found to be pushed to the right, the ascending and transverse colon to be grossly dilated, and the descending colon to be completely flattened out over a large tumour growing from the left lumbar region. A small fæcal fistula connected the descending colon with the abdominal wall, and communicated with a necrotic cavity in the tumour. Extensive peritonitis had obviously extended from this site. Widespread adhesions needed separation before the tumour, the size of a pumpkin, could be removed. There were smaller tumours the size of walnuts in the adjacent tissues.

The liver, kidney, and adrenals were normal. There was a small adrenal rest 2 in. below the right kidney; the bladder and prostate were normal, and the abdominal lymph-glands were not involved. The thorax and cranial cavities were normal.

The actual cause of death was peritonitis and intestinal obstruction.

PATHOLOGY.—Macroscopically the tumour when removed measured 38 cm. × 30 cm. × 20 cm. and weighed 22 lb.* On section it was homogeneously solid except for a few cysts of about 1 cm. in diameter and the necrotic cavity communicating with the bowel. The tumour tissue was white, soft, and arranged in large lobules. There were a few irregular areas of hæmorrhage. The structure of the outrunner growths was similar.

Microscopically the sections consist almost entirely of irregularly directed fasciculi of regularly arranged large spindle cells which are separated by a minimum of fibrils. The nuclei of the cells are plump and oval, almost filling the cell bodies. They are distinctly variable in size and chromicity, are granulated (*Fig. 400*), and often show nucleoli, centrosomes, and mitotic figures. There is an occasional giant cell of tumour type.

FIG. 400.—*Case 4.* Microscopic appearance of tumour, showing granulation of nuclei.

Blood-vessels are scanty and are represented by blood spaces, most of which have a lining of a single layer of endothelial cells.

On the fasciculation and whorling, the shape of the nuclei, and the sparseness of the connective tissue, the diagnosis of myoma was made. With rather more

* The largest yet reported is a fibroma of 25 lb.⁸

fibrous tissue, the resemblance to uterine fibroids would be very close. No cross-striation could be demonstrated in the protoplasm.

The great variation in the size of cell and the mitotic activity of the nuclei together with the immaturity of this very cellular muscle tissue would indicate that degree of malignancy which was evidenced by the autopsy findings.

This tumour is therefore regarded as a leiomyosarcoma. It is possibly because the tumour is myomatous rather than sarcomatous that the malignancy is of local type only, the usual spindle-celled sarcoma of this size in other parts of the body generally having blood-stream metastases.

Case 5.—Retroperitoneal tumour of left iliac fossa.

S. E., female, aged 65. For about four months the patient had noticed a gradual increase in size of her abdomen. For two days she had suffered constant gnawing pain which radiated over the whole abdomen and was accompanied by nausea and much flatulence. The bowels had opened normally. Twelve months previously hysterectomy had been performed for a fibroid, which, though degenerating, was non-malignant by microscopy.

ON EXAMINATION.—There was a large rounded mass palpable in the left iliac fossa. It was mobile and appeared to be attached by a pedicle rising from the pelvis. The tumour was dull to percussion and extended across the mid-line to the right side. The flanks were resonant and vaginal examination elicited nothing of importance.

OPERATION.—Though the nature of the tumour was not known, the abdomen was opened through a lower left paramedian incision for the relief of intestinal obstruction. A large gelatinous mass was found rising from the left side of the pelvis. The exact pedicle could not be ascertained as the tumour 'came away' in soft masses with much bleeding. However, as much of the tumour as possible was removed in this piecemeal fashion with some smaller masses in the omentum. There was very little free fluid, and the intestine was not obstructed.

The wound healed, and after a course of deep X-ray treatment the patient was discharged 'improved'. Six months later her condition was much worse and she was unable to report for treatment. Six months after this she could not be traced, so death had probably supervened.

HISTOLOGY.—In the main, the appearance of the microscopic section of this tumour (*Fig. 401*) is identical with that of *Case 4*. Most of the tissue is just as cellular, but there are numerous areas of necrosis into several of which extensive hæmorrhage has occurred. There was no cystic development. The cells were not quite as atypical and giant cells were rare. This tumour is also regarded as a leiomyosarcoma.

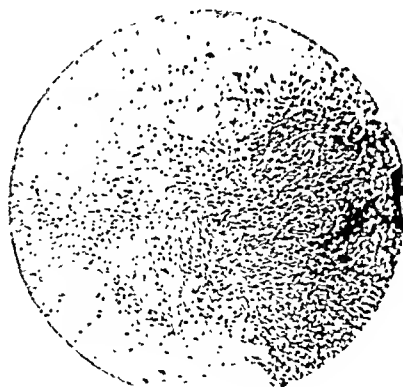


Fig. 401.—*Case 5.*
Microscopic appearance of tumour.

Case 6.—Lumbar retroperitoneal tumour.

M. B., female, aged 64. For four months this patient had suffered dull epigastric pain, worse at night, but unaccompanied by vomiting, nausea, dysuria, or other symptoms. She herself felt the tumour in her abdomen.

ON EXAMINATION.—There was a hard, nodular, immobile mass in the epigastrium the size of a foetal head. Examination of the blood revealed a secondary anaemia.

OPERATION.—At operation through a right upper paramedian incision a large smooth vascular tumour presented. It rose from the posterior abdominal wall and

extended from diaphragm to pelvis, pushing the pancreas and root of mesentery forward. As it was considered inoperable, a portion was excised for section and the abdomen closed.

The patient was discharged improved, and a course of deep X-ray therapy arranged. This patient was not improved by the deep radiation and died within two months of operation.

HISTOLOGY.—At first sight the microscopic appearance of this tumour differs somewhat from that of the others. There are many areas of fat and other areas of tumour tissue in which the cells are much smaller, and the fasciculation is not as definite as in the foregoing tumours. However, there are some areas where the reticulum is arranged in layers and the spindle shape of the cells is visible (*Fig. 402*). Transition from this to the more haphazard arrangement of small ovoid or polyhedral cells in a network of collagen and elastic fibres can be traced.

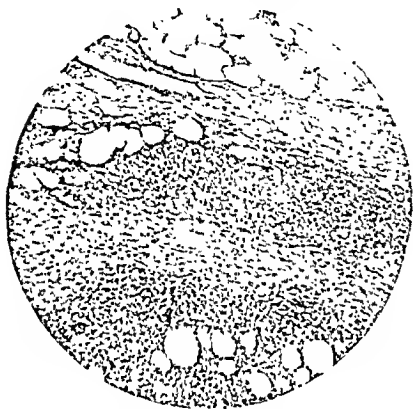


FIG. 402.—Case 6.
Microscopic appearance of the tumour.

The nuclei of the typical cells have pointed ends and are probably fibroblasts, so that the tumour may be regarded on morphological grounds as a fibroma rather than a myoma. It is, however, very difficult to distinguish microscopically a fibromyoma from a cellular fibroma of this type, as the young cell bodies of a cellular fibroma have practically the same staining qualities as the myoma cells. Embryologically in fact they are almost identical cells.

The fatty areas probably represent the remains of the subperitoneal fat, as the section taken at operation could only be from the periphery. They may, however, be neo-

plastic, when the tumour could be better regarded as a mixed tumour of mesoblastic elements.

The fact that there are no grossly atypical cells as appear in sections of *Cases 4* and *5* gives the impression that the tumour is not rapidly growing, though the growing edge shows the infiltration of local malignancy.

DISCUSSION.

Rather less than half the cases in the literature of mesenteric tumours are malignant and are referred to as sarcomata, fibrosarcomata, myxosarcomata, myosarcomata, or liposarcomata. Of the benign form, the lipoma is the commonest 'pure' tumour, and fibromas are quoted as the rarest,⁹ though probably a pure myoma is the rarest. 'Mixed' tumours, however, occur much more frequently than pure tumours—all combinations of fibro-, lipo-, myxo-, myo-, and even chondro- being employed to designate the tumour.¹⁰ Whilst most of the fibromyomata are of the leiomyoma type, a rhabdomyoma type has also been described.¹¹

In this nomenclature, the mesenteric tumours above may be termed a 'fibromyxomyoma' (*Case 1*), and a 'fibromyxoma' (*Case 2*). The retroperitoneal tumours comprise: 1 fibromyxoma, 2 leiomyosarcomata (*Cases 4* and *5*), and 1 fibrosarcoma (*Case 6*). Yet there are identical appearances in some part or other of each by microscopy—compare *Figs. 397, 401, and 402*.

They may be collectively described as spindle-cell tumours, but it would appear that they are really of the type of tumour known as 'mixed'. This, however, is rather a loose term. 'Mixed' parotid tumours, for example, have now been crystallized into epithelial tumours with metaplasias and degenerations and carcinomatous potentialities, and the tumours of this series can be similarly regarded as of one type of original spindle mesoblastic cell with analogous degenerations and metaplasias, and sarcomatous potentialities.

In fact, developing the theme further, it seems that a gradation of embryonal tumours exists. The original embryonic cells were totipotent, but as development and specialization occurred, cells lost certain potencies and became only multipotent and later unipotent. If one could postulate a stranding of these cells in embryonic life in paramesenteric situations with later recrudescence, then a totipotent *Anlage* would produce a teratoma or tridermal tumour; a very multipotent one a bidermal dermoid (e.g., hypoblast and epiblast); a less multipotent one a 'mixed' tumour of one layer only (e.g., a dermoid with epiblastic tissues only, or a mixed paramesenteric tumour with mesoblastic derivature only); and the unipotent one would produce the ordinary simple tumours that we know as fibroma, lipoma, or myoma. Malignancy may supervene at any stage of the evolutionary history of these Rip Van Winkle cells, and the teratoma or mixed tumour or simple tumour become malignant. Tourneux¹² is of opinion that many of the benign paramesenteric tumours become sarcomatous—a belief well borne out by the histological appearances of the above tumours, and the incidence of sarcoma in the literature of this type of growth.

The following case is of a retroperitoneal tumour which must have been frankly sarcomatous from the outset. It is not a spindle-celled or mixed tumour, but is included to show the great variation which may occur when extreme malignancy is the feature, fortunately rarely, of these tumours.

Case 7.—Retroperitoneal sarcoma with metastases.

I. W., female, aged 41. There was a history of six weeks' pain in the left iliac fossa and left thigh. There had been amenorrhœa for four months. During the last six weeks multiple subcutaneous nodules had appeared over trunk and limbs, and the patient became weak and emaciated. In the last two days she had complained of severe occipital headache and vomited continuously.

ON EXAMINATION.—The patient was very ill, but apart from the subcutaneous nodules little could be ascertained. No tumour was palpable in the abdomen. There were no neurological phenomena elicited. The patient died two days after admission.

AUTOPSY.—There were about fourteen of these subcutaneous nodules, and the largest was 1 cm. in diameter. There were numerous small nodules about 4 mm. in diameter scattered through the lung, and several larger ones in the retroperitoneum and omentum. The largest of these was 5 cm. in diameter and overlay the left psoas muscle. The pancreas and liver, spleen, kidneys, adrenals, and intestines were normal. The uterus was carefully examined but was normal. There was a similar tumour in the cerebellum and other smaller ones in the cerebrum.

The ribs, skull, lumbar vertebrae, and sternum were sectioned but were normal, as were the other bones of the skeleton.

HISTOLOGY.—The tissue consists of masses of tumour cells in no definite arrangement. A very fine reticulum or fibrillar mesh separates the individual cells, and in some places there are isolated strands of connective tissue. The cells themselves are exceptionally anaplastic. Most of them are irregularly polyhedral in outline, but not a few are ovoid. The nuclei show excessive mitotic activity.

All degrees from the single immature cell, through the larger more immature cell, to the immature syncytial giant cell formations are well shown (*Fig. 403*). All this betokens a high degree of malignancy which is not belied by the widespread blood-stream metastases, which the autopsy showed to be present in brain, sub-

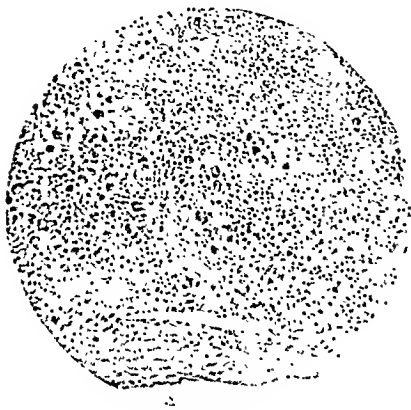


FIG. 403.—*Case 7*.
Microscopic appearance of tumour.

cutaneous tissues, and omentum. Local infiltration can be seen at the periphery of the tumour. Owing no doubt to the rapidity of growth, necrosis occurs in the most ischaemic situations, so that some areas show a peritheliomatous arrangement of tumour cells around the open blood spaces. The vessels are poorly developed and there is a considerable extravasation of blood into the least cellular areas.

The structure of the tumour strongly recalls that of giant-cell tumours of bone, tendon-sheaths, and other mesoblastic tissues, which tumours, be it remembered, whilst generally innocent, may assume malignant characters and metastasize by the blood-stream. There were no tumours in the bony skeleton in this case. The tumour must be regarded as a pure paramesenteric sarcoma, which is so much more anaplastic and malignant than the ordinary

spindle-celled sarcoma that it has almost lost its spindle characteristics and the 'mixed-cell' appearance.

The low grade of malignancy of the tumours can be explained by the lateness of its supervention. They are locally infiltrative, though not to the same degree that carcinomata are; and would probably recur if not completely removed. The earlier the malignancy develops, the more rapidly does local infiltration occur. Metastasis by the blood-stream is also possible then. *Case 20* of the Mayo Series of mesenteric tumours⁶ had secondaries in the liver, and *Case 7* of the above series had secondaries in the brain and subcutaneous tissue, and, as can be seen from the anaplasia of the microscopic section, must have been malignant from the outset.

Actual teratomata, though rare, have been reported¹³—even chorioma.¹⁴ So also have dermoids.¹⁵ All the 'double-barrelled' spindle-celled tumours reported above now fall into one pathological group which can still be called 'mixed' tumours.

It is interesting to note at this juncture that similar mixed tumours,¹⁶ and dermoids, occur in the mediastinum. The original mesenchyme which formed both paramesenteric and mediastinal connective tissues was a happy hunting ground for the early multipotent cells.

One point that arises out of the above is that when dealing with apparently simple tumours, e.g., fibroma or lipoma, it must be remembered that other mesoblastic elements may be present unnoticed in but a cursory pathological examination. Many apparently simple lipomata when removed show fibromatous or sarcomatous tissue in perhaps one spot only,¹⁷ and the infiltrating edges of some simple lipomata can be explained only by the presence of these areas.

There are described other paramesenteric tumours whose appearances are suggestive of a primitive intestinal or renal origin. Most of these, though

not all, are by virtue of their glandular structure cystic. MacAuley¹⁸ reported a series of ileocaecal cysts thought to be enterocystomata, and Higgins and Lloyd¹ report a 'mesenteric' cyst thought to be a pararenal one derived from Wolffian body remains. Some, too, are thought to arise from Müllerian body remains or ovarian rests, and these too are naturally cystic.¹⁹

In recent American literature it is wondered whether mesenteric myomata or fibromyomata are not derived from primitive muscle cells which have wandered away from the uterus or broad ligament. This is not so, of course, as these tumours may occur in the male. Nor do they arise from misplaced cells of the ligament of Treitz or the pelvic fascia; they arise *in situ* from multipotent cells. This question is well dealt with by Carter.¹⁵

Classification of True Paramesenteric Tumours.—One is now in a position to formulate a classification of paramesenteric tumours which may be used both clinically and pathologically:—

BENIGN.—*A. Simple*: Lipoma, Fibroma, Myoma.

B. Embryonal.

1. Teratomatous.
2. Dermoid.
3. Mixed (Cases 1, 2, 3).
4. Developmental—
 - a. Intestinal.
 - b. Wolffian.
 - c. Müllerian.

MALIGNANT.—*A. Simple*: Sarcoma (Case 7).

B. Embryonal.

1. Teratomatous—
 - a. Sarcoma.
 - b. Carcinoma.
2. Sarcoma arising in mixed tumours (Cases 4, 5, and 6).

This classification may also be applied to paramesenteric cysts. The older classification of these cysts was into serous, chylous, sanguineous, echinococcal, and dermoid varieties, and the most recent (Ewing's) into chylous, enteric, nephrogenic, and dermoid.

Serous cysts are degenerative: they may be seen developing in the caseous and myxomatous areas of the tumours of Cases 1, 2, and 3 above. The chylous cysts are of similar causation probably, and the sanguineous ones are hæmatomata only. So that all these may be put in the simple group. Ewing's enteric, nephrogenic, and dermoid cysts then fit the embryonal group of the above classification.

It is not impossible that a certain number of paramesenteric cysts may arise by degenerative processes liquefying the centres of such ill-nourished tumours as that of Case 2.

Theories of Origin of Paramesenteric Tumours.—These may be briefly tabulated: (1) At one time they were supposed to be a form of extra-uterine pregnancy—later it was discovered the tumour could develop in the male! (2) Theory of foetal inclusions—the tumours were compared with parasitic monsters! (3) Theory of parthenogenesis. (4) Theory of heterotopy and cell rests. (5) The embryonal theory as expounded above. The last theory

seems to afford the most satisfactory explanation of the pathological nature and behaviour of the tumours.

Signs and Symptoms: Differential Diagnosis: Treatment: Prognosis.—These questions, while not unimportant, are not as intriguing as the pathology, and have been satisfactorily dealt with by other writers.

The tumours seem somewhat more frequent in the female; Pakowski²⁰ puts the ratio at 70 per cent, and in this series the proportion is 5 females to 2 males. The sexes were equal in the Mayo Series. They may occur at any age, though the malignant ones develop generally after 40.

Signs and Symptoms.—The initial symptom complained of is either the presence of the tumour itself or the onset of a complication: whether the tumour be mesenteric or retroperitoneal, the commonest complications are: (1) Intestinal obstruction; (2) Urinary symptoms; (3) Torsion and degeneration with or without hæmorrhage into the degenerated area.

Of the above 7 cases, 4 noticed the tumour and attributed vague pain to it, 1 complained of urinary symptoms, 1 of swelling of the leg the result of pressure on the iliac veins, and 1 of secondaries in the skin. One case developed intestinal obstruction and a fecal fistula later.

The shortness of history, rapidity of growth, and the presence of secondaries or ascites are suggestive of malignancy. In all the cases there was a definite grade of secondary anaemia present.

The actual signs of the tumour must depend on its site and nature. As a general rule mesenteric tumours are mobile, but neither of the two reported here was—they were too large and adherent. The retroperitoneal ones are generally fixed, but one of these reported above (*Case 3*) was ballotable in the pelvis as it was pedunculated. They may be solid or fluctuant. The flanks are resonant unless a secondary ascites or intestinal obstruction be present.

Diagnosis.—The differential diagnosis is a question of exclusion diagnosis: cysts and tumours of other organs in the region of the tumour must be eliminated clinically or with the help of pyelogram, cholecystogram, and barium meal or elysma. The blood should be examined for evidence of leukaemia, the Casoni and complement-fixation tests performed for hydatid elimination, Wassermann reaction taken, and Hodgkin's disease excluded.

It should be noted that in ordinary experience the commonest cause of a spherical hard abdominal tumour which can be moved into any quadrant of the abdomen of an adult over 20 is not generally a mesenteric cyst or tumour but a carcinoma of the colon, or, less commonly, calcified mesenteric glands.

Exploration is advisable, as complications and malignancy occur, and in any case pre-operative diagnosis is always a little uncertain.

Treatment.—The tumour should be excised if this is possible. If its relationship to other structures such as the pancreas or intestinal blood-vessels is too intimate, or there is too much infiltration, there is nothing further to be done. In excising a tumour in the mesentery of the bowel, the circulation of the corresponding loop must be observed. Resection is often necessary.

In inoperable cases deep X-ray therapy may be tried. In *Cases 5* and *6* above there was no improvement whatsoever with deep X-ray. This affords

a striking contrast to the behaviour of lymphosarcoma under deep X-ray treatment.

Prognosis.—In the matter of prognosis, which has not been fully discussed by previous writers, the following points emerge: (1) Only about one-third of these tumours can be removed at operation; in this series 3 out of 7 were removed. (2) The immediate mortality rate is about 40 per cent.²¹ (3) When removed completely, the prognosis is good once the patient gets over the shock; however, only one case in this series of 7 is alive three years after operation. (4) An idea of the malignancy of the tumour may be obtained from a study of the histological appearance of a section and a prognosis given on that. (5) Deep X-ray treatment does not improve the prognosis.

CONCLUSIONS.

1. A series of 2 solid mesenteric tumours and 4 retroperitoneal tumours are reported. They are classed together as paramesenteric tumours.

2. The similarity of their pathology is discussed and their embryonal origin suggested.

3. A classification is put forward suitable for both solid and cystic tumours.

4. The signs, symptoms, diagnosis, treatment (including X-ray), and prognosis are briefly discussed.

I am indebted to the honorary surgical staff of the Alfred Hospital for permission to publish these cases.

REFERENCES.

- ¹ HIGGINS and LOYD, *Brit. Jour. Surg.*, 1924, xii, 95.
- ² HARRIS and HERTZOG, *Ann. of Surg.*, 1897, xxvi, 66.
- ³ VANCE, *Ibid.*, 1906, xliii, 366.
- ⁴ GREER, *Brit. Med. Jour.*, 1911, ii, 1085.
- ⁵ SZENES, *Deut. Zeits. f. Chir.*, 1918, exliv, 228.
- ⁶ RANKIN and MAJOR, *Surg. Gynecol. and Obst.*, 1932, May, 809.
- ⁷ MILLAR, McW., *Brit. Jour. Surg.*, 1928, Oct., 323.
- ⁸ DARNALL, *Ann. of Surg.*, 1928, lxxxvii, 870.
- ⁹ JUDD and McVAY, *Surg. Gynecol. and Obst.*, 1920, xxxi, 372.
- ¹⁰ DICKENSON. BRICHETEAU. Included in list of Harris and Hertzog.
- ¹¹ CONFORTI, *Centrab. f. allgem. Pathol.*, 1906, xvii, 817.
- ¹² TOURNEUX, *Presse méd.*, 1929, xxvii, 1014.
- ¹³ BLAND-SUTTON, *Tumours Innocent and Malignant*, 505.
- ¹⁴ BONNEY, "Omental Chorioma", *Trans. Pathol. Soc. Lond.*, 1907, lviii, 9.
- ¹⁵ CARTER, *Surg. Gynecol. and Obst.*, 1921, xxxiii, 544. Specimen 1681, Melbourne University Pathological Museum.
- ¹⁶ ROBINS, *Amer. Jour. Roentgenol.*, 1929, xxii, 351; ROBERTSON and BROWN, *Brit. Jour. Surg.*, 1929, xvii, 197.
- ¹⁷ EWING, *Neoplastic Diseases*, 3rd ed., 1928, 190.
- ¹⁸ MACAULEY, *Brit. Jour. Surg.*, 1923, xi, 122.
- ¹⁹ DOWD, *Ann. of Surg.*, 1900, xxxii, 515.
- ²⁰ PAKOWSKI, *Arch. gén. de Chir.*, 1912, viii, 1029.
- ²¹ ROYSTER, *Jour. Amer. Med. Assoc.*, 1911, lxxii, 534.

OBSERVATIONS ON PLEURAL ABSORPTION.*

By R. C. BROCK,

SURGICAL REGISTRAR, OUY'S HOSPITAL, AND CONSULTING THORACIC SURGEON TO THE L.G.C.

THE mortality of the so-called post-pneumonic, usually pneumococcal, type of empyema is with even moderate skill in treatment a low one. In the so-called syn-pneumonic type, which is usually streptococcal in origin, the state of affairs is quite different, and it is generally accepted that even with the most skilful treatment the mortality rate is rarely less than 10 per cent, and, depending upon the severity of the particular epidemic, frequently higher. It is true that in many cases of this type death actually occurs from some concurrent major condition such as septicæmia, pneumonia with abscess formation, or meningitis, and that the empyema may be merely an incident in the course of an overwhelming general infection, but there can also be little doubt that the presence of a suppurative inflammation of such a large serous membrane as the pleura must have a seriously adverse effect on the course of the illness. Just as it is a mistake to attribute the whole of a patient's desperate condition to the suppurative pleurisy while the presence of an extensive underlying pneumonia is overlooked, so is it a mistake to overlook the fact that the additional burden of an indifferently treated pyothorax may turn the balance against the patient.

In spite of this admitted persistent high mortality, no new suggestions for treatment have been made during the last fifteen years—that is, since the recommendation of aspiration in the early formative stage and postponement of rib resection until the pneumonic condition has subsided. It is clear that before any logical improvement in treatment can be suggested it is essential to know more of the underlying physiological and pathological processes taking place in the pleura, and of these processes it seems that of first importance is absorption. Absorption from this large serous membrane must be the main feature causing the illness resulting from its infection. This illness arises partly from the absorption of bacterial and other toxins and partly from absorption and dissemination of micro-organisms themselves. There is ample experimental and other evidence to show that bacteria are freely absorbed from the pleura into the general circulation. Wadsworth,¹ Corfer,² Higgins and Lemon,³ and other workers have all shown that particulate matter such as carmine and indian ink or bacteria is readily absorbed. Thus Higgins and Lemon,⁴ after studying the results of injection of finely-divided graphite particles, state: "The sternal and the tracheal lymph nodes were always densely black within one hour. It was obvious, then, that the lymph drainage occurred promptly and that free particulate matter was

* Read at a meeting of the Association of Surgeons in Manchester, May, 1933.

distributed into the lymph nodes together with that contained within the phagocytic cells both of the polymorphonuclear and the monocytic types. Furthermore it appeared that the material absorbed and transferred through the lymphatics reached the thoracic duct or the right lymphatic duct and emptied into the venous circulation within a period of approximately one hour; it thus became possible for such particulate material to reach the systemic circulation and to appear in the liver, spleen and bone-marrow within a relatively short time after injection. . . . Within two to three hours of the injection the Kupffer cells were swollen to several times their original size, and they encroached on the lumen of the sinusoids, or desquamated into the lumen. This appearance always preceded the recognition of particulate matter within them.' This proves quite definitely that an open pathway exists for particulate matter from the pleura into the general blood-stream.

It is to be noted that no attempt is made in this paper to add further arguments to the discussion that exists as to the relative parts played by the lymphatic system and blood-vessels in the removal of exudate from the pleura. The generally accepted view is probably the correct one—namely, that removal of particulate material, including bacteria, takes place by way of the lymphatics, and soluble material by way of the blood-stream. Starling's work⁵ seems to prove this latter completely, and the work of the other investigators already mentioned amply supports the former. It is probably not out of place to draw a comparison with an infection of the cellular tissues of the hand or fingers; the infection may remain strictly localized with but minor symptoms of general disturbance from toxæmia; or it may spread to the axillary lymph-glands, with or without an obvious lymphangitis; or it may lead to general blood-stream infection with great prostration or death of the patient. It is not suggested that in every case of pyogenic infection of the pleura general dissemination of micro-organisms takes place; it is clearly the function of the lymphatic system to seize, and if possible fix, the invading bacteria, and there is but little doubt that this fixation in the lymph-vessels and lymph-nodes of the mediastinum³ is successful in the majority of cases, just as all but very few infections of the cellular tissue of the hand are effectively prevented from spreading beyond the axillary lymph-nodes. No one will deny, however, that when the pleura is infected there is a much greater tendency for general blood-stream infection to occur, either on account of low resistance on the part of the patient (e.g., infants) or of unusual virulence on the part of the infecting organism (e.g., epidemics). For the purpose of this paper it is taken for granted that the absorption of the purulent fluid is harmful; and it is also implied that while soluble toxins can be absorbed, so can micro-organisms. Indeed, one of the aspects which it is wished to stress is that this absorption of bacteria and toxins from the infected pleura may be the most prominent feature in the causation of the secondary—septicæmic—manifestations from which death so often occurs.

Although, as already mentioned, much work has been done on the absorption of particulate matter from the pleura, almost no investigations have been made into the factors governing the absorption of fluids by the pleura, except for some observations by Starling, Leathes, and Tubby^{5, 6} nearly

forty years ago. This is the more remarkable when one considers the important part the pleura plays in practical medicine and surgery.

The animals used in my investigations were rabbits. These have the disadvantage of possessing a relatively small chest as compared with the dog, but the conditions in the rabbit's chest are much more comparable to those in the human owing to the more stable and relatively impermeable mediastinum it possesses, whereas the dog's mediastinum readily allows passage of air and fluids from one side of the chest to the other. The experiments were conducted under intravenous nembutal anaesthesia, a method of great value in observations on the respiratory apparatus seeing that one is able to eliminate the difficulties associated with inhalation anaesthesia. The general plan has been to introduce fluid into the chest by means of a fine cannula from a burette, and at the end of the experiment to open the chest and suck out the remaining fluid, which was then measured in the same burette.

ABSORPTION FROM THE UNINFLAMED PLEURA.

The simplest fact that has been ascertained is, as could almost have been predicted, that the rate of absorption of fluid, i.e., normal saline, varies directly with the volume of fluid present. Thus, it is slow with a few cubic centimetres of fluid and rapidly increases as the volume grows larger. *Table I* shows the increase of rate of absorption of normal saline (1) with increase of volume of fluid in the chest, (2) with increased depth of respiratory movements.

Table I.—SHOWING INCREASE OF RATE OF ABSORPTION OF NORMAL SALINE.

VOLUME OF FLUID	VOLUME ABSORBED	
	Shallow Respiration	Deep Respiration
c.c. 20	c.c. 0.75	c.c. 3.0
50	4.25	7.5
75	7.50	10.5
100	7.25	14.0

This is also shown by the varying times of survival of animals after the intrapleural injection of varying quantities of a strychnine solution in normal saline. Thus, if a solution is made up containing a lethal dose in 5 c.c. of fluid and only 5 c.c. are introduced into the pleura, death does not occur before some twelve hours. On the other hand if 50 c.c. of the same strength solution are given, death occurs much more rapidly—in fact in two hours.

The importance of variation in the respiratory movements has next been studied, and the results are striking. An account of some initial experiments on this aspect of the importance of the respiratory movements was published in an earlier paper,⁷ but since then fuller investigations have been made.

With quiet respiration the rate of absorption is moderate; as the respiratory movements become deeper and more laboured (for example, following the

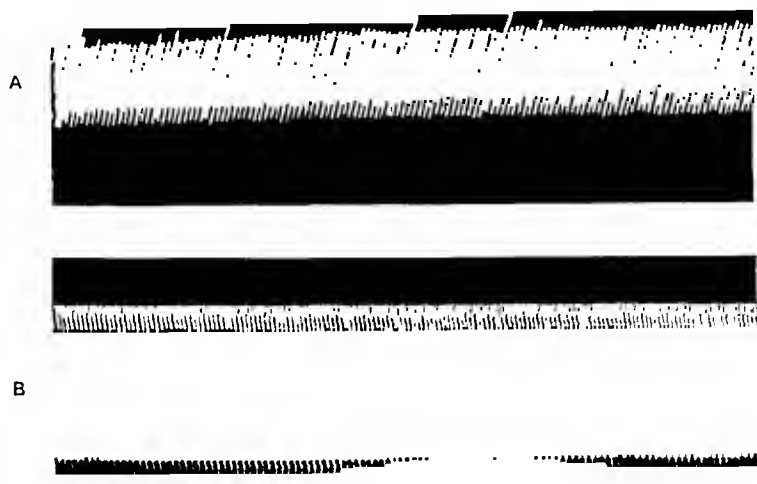


FIG. 404.—Tracings of respiratory movements from two rabbits after the introduction of 50 c.c. of normal saline into the pleura. The animal breathing deeply (A) absorbed 9.6 c.c. in one hour; the one breathing quietly (B) absorbed only 5 c.c. in the same time.

administration of CO_2 or partial occlusion of the trachea) the rate of absorption increases. *Table I* shows this quite clearly and also shows the variation in the absorption rate with variation of the volume of fluid in contact with the pleura. (See also Fig. 404.)

In other words, if a small amount of fluid is in the chest and respiration is not embarrassed, very little absorption takes place. If, however, a maximal amount of fluid is present and respiration is laboured and deep, then absorption occurs at a surprisingly rapid rate. This is even better shown in the dog when an animal with 200 c.c. of saline in the pleura and breathing quietly absorbed 15 c.c. in two hours, and a similar animal breathing forcefully absorbed 139 c.c. (Fig. 405).

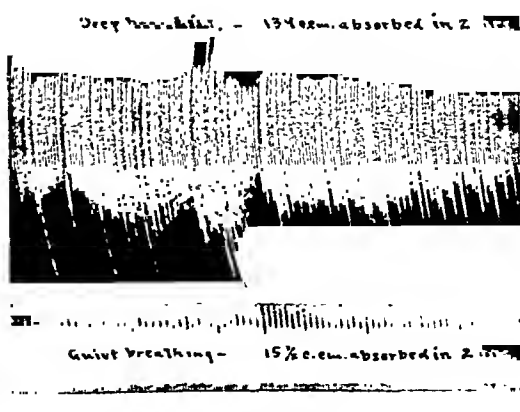


FIG. 405.—Simultaneous tracing from two dogs after the introduction of 200 c.c. of normal saline into the pleura. The animal breathing deeply (upper part of figure) absorbed 139 c.c. in 2 hours; the other animal, breathing quietly, absorbed only 15½ c.c. in the same time.

The importance of the respiratory movements can also be shown dramatically by comparing the survival time of two rabbits of the same litter after the introduction into the pleura of 50 c.c. of strychnine solution. A cannula in the opposite pleura connected with a tambour was used to give the record shown in *Fig. 406*. The animal which breathed quietly survived twice as long as the animal whose depth of respiration was increased by administering CO_2 , and in each case on aspirating the fluid remaining in

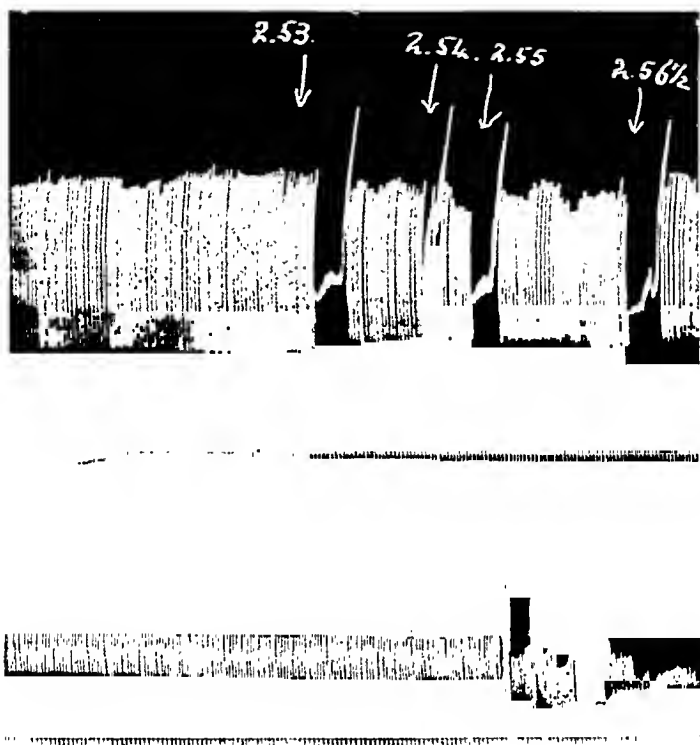


FIG. 406.—Tracings of respiratory movements from two rabbits of the same litter after introduction into the pleura of 50 c.c. of saline containing strychnine. The upper curve is from the one to which CO_2 was administered, and death occurred in 35 minutes. In the lower curve breathing is of average depth, and death did not occur before 55 minutes. The arrows pointing to interruptions in the tracings mark convulsions.

the chest it was found the approximate volume containing a lethal dose had been absorbed, i.e., 5.5 c.c. in the case of the animal breathing deeply, and 4.85 c.c. in the case of the other; this gives an hourly rate of 9 c.c. and 5.4 c.c. in each case. In a similar experiment with a weaker solution of strychnine the times of survival were 135 minutes with quiet respiration and 70 minutes with deep respiration.

The increased rate of absorption with greater volume of fluid probably results from the greater surface area in contact with the fluid. The increase

with deeper respiratory movements may, of course, be partly due to the greater agitation giving a wider surface of contact, but this cannot be the sole factor, and it is probable that the chief reason is that the increase of power in the respiratory pump causes a greatly increased circulation of both blood and lymph.

ABSORPTION FROM THE INFLAMED PLEURA.

The question that next arises is whether the absorption rate is at all modified by inflammation of the pleura. The only work having any direct bearing on the subject I have been able to discover is the observation by Starling⁶ that the absorptive power of the pleura could in no way be affected either by scalding the lining with hot water or by treatment with sodium fluoride, and Corfer's experiment² on the removal of particulate matter following treatment of the pleura with dilute HCl. from which he concluded that the absorption rate is delayed.

Fluid accumulates in any serous cavity when the balance between the rate of formation and absorption is altered. No one will deny that when the pleura is inflamed fluid accumulates chiefly because it is formed at a greatly excessive rate. The result of the inflammation on the rate of absorption is, however, masked by this great outpouring of fluid, but a moment's consideration will show that it can either remain unaltered, can be diminished, or can be increased. It is, moreover, of considerable practical importance to know the exact change, if any, that takes place in the rate of absorption, and it is indeed a surprising fact that this question has so far been given such scant attention and that no one has attempted to settle the problem definitely. It has been accepted that absorption does occur, but whether at an increased, decreased, or unaltered rate is not known. Some, in fact, have gone so far as to suggest that no appreciable absorption can occur when exudation is occurring, for fluid can scarcely pass both ways to and from blood-vessels and lymphatics. This latter argument, however, entirely overlooks the phasic conditions resulting from the respiratory movements which undoubtedly play a very important part in absorption.

It was found that the simplest and most certain way of producing as it were a standard inflammation of the pleura was by the injection of a few minims of turpentine. Within twenty-four hours an intense sero-fibrinous reaction occurs with formation of a large effusion. If the injection is repeated after a day or two, this progresses to the stage of thick pus formation in a well-shut-off empyema cavity.

The problem that presents itself is the difficulty of accurately determining the volumetric absorption of fluid from a cavity into which exudation has occurred and is still occurring. It is clear that, however carefully one aspirates the chest dry at the beginning of the experiment, one can never be sure that some fluid is not left in (in fact one might say that one can be sure that some fluid will be left in) and also fluid is being formed at an unknown rate during the actual experiment. These difficulties have been circumvented, at any rate in part, by the use of a saline solution of strychnine containing a lethal dose of the drug in a known volume of the fluid. After

aspiration of the existing exudate this solution is injected into the pleura and the death of the animal gives a definite end-point. The fluid remaining at the end of the experiment is measured.

Using this method, the absorption rate has been estimated at stages of pleurisy ranging from twenty-four hours to eighteen days, and the results have been definite and constant in showing an increased rate of absorption. This increase is more marked in the earlier stages (twenty-four to forty-eight hours). *Table II* shows results from these experiments, and it can be seen that a control animal would survive 100 minutes with absorption of the requisite amount of fluid containing a lethal dose, while the animal with an inflamed pleura would survive only 35 minutes. Even in the third week the absorption rate is at least doubled.

Table II.—SHOWING INCREASED RATE OF ABSORPTION WITH ACUTE INFLAMMATION AND INCREASED DEPTH OF RESPIRATION.

DAYS OF INFLAMMATION	TIME OF SURVIVAL	CONTROL
SHALLOW RESPIRATION		
	Mins.	Mins.
24 hours	35	100
3 days	25	180
4 days	53	180
10 days	60	135
15 days	69	120
DEEP BREATHING		
24 hours	25	100
48 hours	15	135
5 days	40	180
18 days	25	120

The amount of fluid absorbed by the end of the experiment in the animals with an inflamed pleura varied, as one would expect, since it was not possible to be sure that one had aspirated all the exudate at the beginning of the experiment and fluid was also being formed during the whole course of the experiment. In spite of this, volumetric confirmation of the increased rate of absorption was frequently obtained in addition to the diminution in the time of survival. Thus :—

R. 47.—Control. Weight 2.6 kilos.

L.D. = $2.6 \times 0.08 = 2.08$ mgrm. (contained in 10 c.c.).

Strychnine solution used = 10 mgrm. in 50 c.c. of normal saline.

12 noon.—50 c.c. of fluid introduced into left pleura. Respiration (controlled by kymographic tracing), average depth.

1.40 p.m.—Death occurred; i.e., in 100 minutes.

Post-mortem.—13.25 c.c. absorbed, i.e., 7.8 c.c. per hour.

R. 48.—Twin of control. Weight 2.6 kilos.

Twenty hours' inflammation; other details identical with control.

2.54 p.m.—50 c.c. of solution introduced into left pleura. Respiration (kymographic tracing) comparable to that of control.

3.30 p.m.—Death occurred, i.e., in 36 minutes.

OBSERVATIONS ON PLEURAL ABSORPTION 657

POST-MORTEM.—14.15 c.c. absorbed, i.e., 24 c.c. per hour. There was a typical diffuse pleurisy with much recent exudate extending over the whole of the left lung and left pleural space.

Comment.—Death occurred three times as quickly as in the control of the same litter and there was close verification in the volume of fluid removed (13.25 c.c. in R. 47, 14 c.c. in R. 48). In both cases, as in all others, the rapid onset of rigor mortis characteristic of strychnine poisoning occurred.

Again :—

R. 74.—Control. Weight 3 kilos.

L.D. = 2.4 mgrm., and this is contained in 12 c.c. of solution.

10.48 a.m.—50 c.c. of fluid introduced into left pleura.

12.44 p.m.—Death occurred, i.e., in 2 hours.

POST-MORTEM.—12.75 c.c. absorbed, i.e., 6.37 c.c. per hour.

R. 73.—Twin of control. Weight 2.9 kilos.

In this case turpentine was injected into the left pleura on two occasions and turbid fluid aspirated at frequent intervals until thick pus was obtained. The final experiment was carried out on the fifteenth day.

Aug. 3.—Aspiration of chest resulted in 32 c.c. of purulent fluid with thick flocculi.

11.13 a.m.—50 c.c. of fluid introduced into left pleura.

12.22 p.m.—Death occurred, i.e., in 69 minutes.

POST-MORTEM.—12 c.c. absorbed, i.e., 10.5 c.c. per hour. There was an intense diffuse pleurisy on the left. The right side of the chest was normal and contained no fluid.

Comment.—The shorter time of survival is confirmed almost exactly by the volumetric absorption.

Such striking confirmation was not always obtained, but there was never an excessive difference between the amount absorbed from the control and from the animal with the pleurisy, and the character of the fluid removed at the end of the experiment in the latter case always showed clear evidence of having been mixed with purulent exudate.

The same variation of rate of absorption with variation in respiratory movements was also shown to occur, i.e., if deep breathing was induced, the absorption rate was increased still further (*see Table II*).

There is an obvious criticism to this method—namely, that the animals die sooner because they are not well; i.e., the lethal dose for them is smaller than for a normal animal. Apart from the confirmatory evidence of the volume of fluid absorbed, as shown above, this objection can also be disproved in another way; if the solution is placed in the opposite, i.e., the healthy, side of the chest, it can be shown that the lethal dose and time of survival are the same as for a healthy animal.

Thus :—

R. 84.—Weight 3.05 kilos.

L.D. = $3.05 \times 0.8 = 2.44$ mgrm. (= 12 c.c. of fluid).

July 30.—Injection of turpentine into left pleura (ether anaesthesia).

Aug. 3 (fourth day).—Aspiration of left pleura resulted in removal of 17 c.c. of turbid fluid.

11.45 a.m.—50 c.c. of fluid into *right* side of chest. Respiration of average depth.

3.20 p.m.—Death occurred, i.e., in 3 hours, 35 minutes.

POST-MORTEM.—14 c.c. of fluid absorbed from the right chest, i.e., 3 c.c. per hour. On the left side there was a diffuse acute pleurisy and 6 c.c. of turbid fluid were present, i.e., either this amount had not been removed at the preliminary aspiration, or it had formed during the course of the experiment, and in any case illustrates well how unsafe it would have been to rely on volumetric methods alone to estimate absorption from the inflamed side.

R. 95.—Weight 3.2 kilos.

Aug. 25.—Injection of turpentine into left chest.

Sept. 3.—Ninth day of pleurisy. Preliminary aspiration gave 35 c.c. of purulent fluid from the left pleura.

10.38 a.m.—50 c.c. of fluid into *right* chest. Respiration of moderate depth.

12.43 p.m.—Death occurred, i.e., in 125 minutes.

POST-MORTEM.—13.5 c.c. absorbed from right pleura (L.D. = 12.8 c.c.). Left pleura showed splendid example of suppurative pleurisy with 3 c.c. of fluid present.

It is not suggested that this strychnine method of estimating the absorption rate from the inflamed pleura is without fault. It is only intended to present the method, state the results obtained from it, and draw certain conclusions in view of the consistently similar results. It is felt that in the absence of a better method the observations are worthy of notice. Although in each case an approximate lethal dose has been worked out, it must be made quite clear that such an arbitrary dose alone is of no true scientific value; apart from other considerations, the weight of the animal may be inaccurate, e.g., from a recent meal or from rapid wasting. For this reason it has been a constant practice to use as a control an animal of the same litter which was also in apparent good health.

The objection that one is using a crystalloid toxic substance and not a colloidal one is more pertinent and is difficult to answer unless one has actually repeated the experiments with a colloidal toxin. To do this, however, would be no light task and would probably introduce other and more complicated sources of error. At the same time I should like to point out that when normal saline is introduced into the pleura, proteins are rapidly added to it, as shown by Starling,⁵ and the absorption rate of this colloid-containing fluid continues unchanged. Some experiments were also carried out during this series in which rabbit's serum was introduced into the pleura instead of normal saline, and it was shown that the same variations of the absorption rate occur, although the whole process is slower with the serum than when normal saline is used.

From the experiments on the uninflamed pleura it was pointed out that a patient with a large effusion and breathing deeply is absorbing fluid from the pleura at a very rapid rate. From these observations in the presence of inflammation it is suggested that in a patient with an acutely inflamed pleura containing a large amount of free fluid and who is correspondingly dyspnoic (or even more grossly dyspnoic from an associated pneumonic process) the rate of absorption is grossly—in fact, one may even say alarmingly—increased. Just as these experimental animals with an acutely inflamed pleura and very dyspnoic killed themselves more quickly by means of the strychnine they absorbed, so it is suggested that the grossly dyspnoic, perhaps cyanosed, patient with a suppurative pleurisy is helping to kill himself more quickly by a more rapid absorption of toxins.

When one remembers the intense hyperemia of acute inflammation, especially during the early stages, it is quite reasonable to suppose that such an increased rate of absorption does occur, and it is suggested that this increased vascularity is the mechanism responsible for the increase.

EFFECT OF PNEUMOTHORAX ON THE ABSORPTION RATE.

When this greatly increased rate of absorption became apparent the obvious step was to attempt to find some further means of diminishing the rate of absorption, and to this end the effect of artificial pneumothorax was investigated. Although there has been a great deal of work on the absorption of gases from an artificial pneumothorax, no attempt has been made to ascertain the effect of air replacement on the absorption of fluids from the pleura. It was expected and hoped that the absorption rate would be greatly diminished when air was added, but this was found not to be so. The rate of absorption was at least doubled. Thus, in *Table III*, the effect of a

Table III.—SHOWING INCREASE OF VOLUME ABSORBED WITH ADDITION OF AIR.

AIR	EFFECT OF PNEUMOTHORAX	
	Normal Saline	Volume Absorbed in 1 Hour
c.c.	c.c.	c.c.
0	25	0.25
45	25	3.25
50	25	3.75

pneumothorax on the volume of absorption is seen, and in *Tables IV* and *V* the effect on the time of survival after using a strychnine solution is illustrated. In these latter experiments the volumetric increase of fluid absorbed also corresponded. As the proportion of air increases so does the rate of

Table IV.

AIR	EFFECT OF PNEUMOTHORAX		
	Volume of Fluid	Time of Survival	Weight
c.c.	c.c.	Mins.	Kilos.
0	50	65	3.0
0	50	54	3.0
25	50	28	3.3
25	50	27	3.25
50	50	30	4.0

absorption. *Table V* shows that deep and laboured respiration further increases the rate of absorption. In other words, absorption takes place more rapidly from a hydropneumothorax than from a hydrothorax, and more

rapidly still when dyspnœa is present. Similarly, when inflammation is present the addition of air still further increases the rate of absorption (*Table VI*). A solution of strychnine in normal saline was employed in these experiments.

Table V.

AIR	EFFECT OF PNEUMOTHORAX		
	Volume of Fluid	Time of Survival	Weight
c.c.	c.c.	Mins.	Kilos.
0	25	180	3.0
10	25	110	2.95
15	25	95	2.7
20	25	88	3.0
25	25	80	3.0
25	25	68	3.45
40	25	66	3.2
50	25	65	3.3
50	25	51	3.15
70	25	38	3.6
DEEP BREATHING WITH CO ₂			
25	25	33	3.2

Once more to recapitulate: the patient who is grossly dyspnœic with a large pyo-pneumothorax and an acutely inflamed pleura is absorbing harmful substances at an alarmingly rapid rate.

The explanation of this increased rate of absorption in the presence of a pneumothorax is difficult to see; it is the reverse of what one would expect, but the results of these observations are so striking and so consistent that only the one conclusion can be drawn from them. It may be stated here that all the series of experiments recorded in this paper have been confirmed at least two or three times, although to avoid confusion only the simplest outline of figures has been given.

Table VI.

AIR	EFFECT OF PNEUMOTHORAX AND INFLAMMATION			REMARKS
	Volume of Fluid	Time of Survival	Weight	
c.c.	c.c.	Mins.	Kilos	
0	50	120	3.0	Control. No inflammation
0	50	73	3.25	Control. 24 hours' inflammation
25	50	50	3.25	Pneumothorax and 27 hours' inflammation

The only other experimental work on the effect of a pneumothorax on absorption from the pleura has been concerned with the fate of particulate matter (e.g., indian ink), and the somewhat meagre results are in direct conflict with those recorded above. Dolley and Wiese⁸ conclude from observing the rate of flow from the thoracic duct that the rate of absorption of trypan blue from the pleura is delayed: they do not seem, however, to be acquainted with Starling's work⁵ in which he showed that the removal of such a dye from the pleura occurs almost entirely by way of the blood-stream, and its appearance in the lymph from the thoracic duct can be accounted for by its filtration from the blood into the tissue spaces and thence into the lymphatic system secondarily. He showed, for instance, that it could be readily demonstrated in the urine at once; Dolley and Wiese make no mention of the urine examination, and one is forced to discount this part of their observations. The well-known experiments demonstrating the greater time taken for the removal of soot particles from the lung on the same side as a pneumothorax was present, although showing undoubted diminution of absorption from the lung, are not applicable to absorption from the pleura.

DISCUSSION.

The practical application of these findings is straightforward, and in pleural suppuration applies especially to the early, so-called formative, stage, when purulent fluid is first noticed in the chest but it has not thickened to form pure pus and the patient is still desperately ill from the accompanying pneumonic process or general infection. It is at this, the most dangerous, stage of the illness that refinements of detail of management of the effusion are lacking. It is clear that aspiration should be repeatedly practised to diminish the total volume of fluid, and all other possible measures taken to relieve dyspnoea. The frequency and need for aspiration I feel is a matter of first importance. At present it is a common practice to aspirate the fluid as often as appears necessary to relieve gross dyspnoea. This usually means fluid is allowed to accumulate for some forty-eight hours, or even longer, by which time the effusion is sufficiently large to cause gross displacement of the mediastinum. Removal of all this accumulated fluid at one aspiration is not only dangerous but frequently impossible owing to the symptoms of distress which rapidly develop, especially in little children. We know from these observations that such a patient has been absorbing toxic substances at an excessive rate; that he has been helping to kill himself more quickly just as the experimental animals did. It is certain that aspiration should be practised more often both to prevent accumulation and to give the maximum relief from dyspnoea. I feel that twice a day is not too often in a really ill patient, and this can be carried out with very little trouble if the step suggested by Tudor Edwards is followed. When the local anæsthetic is administered for the first aspiration a short incision is made down to the intercostal space, and after aspiration the small wound is packed and left open. Subsequent aspirations can be performed with very little disturbance to the patient and with but little fear of a spreading cellulitis of the chest wall.

In the acute stage in which absorption of exudate is not desirable, air replacement, if performed at all, should be minimal and only sufficient to relieve symptoms of distress following aspiration. If aspiration is repeated as often as recommended it is doubtful if symptoms of distress due to removal of fluid should occur. It as to be noted (*see Tables III and IV*) that if air is *added to* fluid in the chest, e.g., 25 c.c. of air added to 25 c.c. of fluid, the absorption rate is increased (survival time after strychnine diminished from 180 to 80 minutes). If, however, air is made to replace fluid in the chest, e.g., 50 c.c. of fluid half replaced by air, the absorption time is slightly diminished (survival time 65 minutes becomes 80 minutes). In other words, the *replacement* of fluid removed by a smaller quantity of air will do but little harm; *addition* of air, or replacement by a larger quantity, will hasten absorption.

There are times, however, when it is wished to assist absorption of an effusion, such as in some cases of clear effusion, and in certain selected cases of empyema. Replacement of the aspirated fluid by air will help absorption, and, where there is no contra-indication (e.g., tuberculosis) the administration of CO₂ at regular intervals will also help. The air replacement of pleural fluids is, of course, no new procedure. It has been strongly advocated by many as a means of treatment. These new observations do serve, however, to put the method on a sound physiological and pathological basis.

SUMMARY.

1. The necessity for a greater knowledge of the underlying physiological and pathological processes occurring in the pleura in acute inflammation is pointed out, and it is suggested that of these processes the most important is absorption.

2. From experimental observation it is demonstrated that absorption of fluid from the pleura varies directly with the volume of fluid present, and, moreover, is profoundly modified by variations in the depth of the respiratory movements. With deep breathing the rate is much increased over that when respiration is moderate or slow in depth.

3. Acute inflammation produces a marked increase in the rate of absorption of fluid.

4. The presence of air in the chest in addition to fluid still further increases the rate of absorption; if inflammation is present and the patient is dyspnoic, as in the case of a large pyo-pneumothorax, the rate of absorption is alarmingly increased.

5. Certain practical conclusions are drawn from these findings.

The greater part of this work was carried out by the writer as Surgical Research Scholar to the Association of Surgeons (1932-3); a smaller portion as the Astley Cooper Student, Guy's Hospital.

I wish to acknowledge with grateful thanks the facilities for work in the Department of Physiology, Guy's Hospital, accorded me by Professor M. S. Pembrey, and the very kind help and advice I received from him. *Fig. 405* is published by permission of the Editor of the *Journal of Thoracic Surgery*.

BIBLIOGRAPHY.

- ¹ WADSWORTH, T. W., *Jour. of Pathol. and Bacteriol.*, 1922, xxv, 266.
- ² CORFIER, H. J., *Jour. Amer. Med. Assoc.*, 1926, lxxxvi, 1739.
- ³ HIGGINS, G. M., and LEMON, W. S., *Amer. Jour. Med. Sci.*, 1931, clxxxi, 697; LATER, J. C., *Ibid.*, 1924, clxviii, 882; KARSNER, H. T., and SWANBECK, C. E., *Jour. Med. Research*, 1920, xlii, 91.
- ⁴ LEMON, W. S., and HIGGINS, G. M., *Amer. Jour. Med. Sci.*, 1932, clxxxiv, 846.
- ⁵ STARLING, E. H., and TUBBY, A. H., *Jour. of Physiol.*, 1894, xvi, 140.
- ⁶ STARLING, E. H., and LEATHES, J. B., *Ibid.*, 1895, xviii, 106.
- ⁷ BROCK, R. C., and BLAIR, E. A., *Jour. of Thor. Surg.*, 1931, i, 50.
- ⁸ DOLLEY, F. S., and WIESE, E. R., *Arch. of Surg.*, 1929, xviii, 542.

THE PLAN OF THE VISCERAL NERVES IN THE LUMBAR AND SACRAL OUTFLOWS OF THE AUTONOMIC NERVOUS SYSTEM.

BY HUGH C. TRUMBLE,

FROM THE BAKER MEDICAL RESEARCH INSTITUTE, MELBOURNE.

For some years past surgeons have displayed great interest in the sympathetic nervous system. Papers dealing with the subject from different aspects have been published in astonishing numbers. It was soon apparent that many of the authors had not a sound conception of the anatomy and physiology of the system, and that the nomenclature was not all that could be desired. Many facts which had been discovered long ago by physiologists were re-discovered by surgeons, and not always did the latter appear to know of or acknowledge the work of the former. The conclusions of distinguished physiologists have been attacked by surgeons who thought that they had discovered facts hitherto unknown, although a careful search through the literature would have shown that the physiologists concerned were not ignorant of the facts and that they had not neglected them in drawing their conclusions. Sweeping assumptions have been made from quite insufficient and ill-established observations.

In connection with the autonomic nervous system an immense amount of careful detailed work has been done by physiologists, who succeeded years ago in presenting a fairly definite though incomplete plan of the nerves. J. N. Langley in particular stands out as a gifted and devoted worker in this field.

For a detailed description of the nerves dealt with in this paper, as seen in cats and rabbits, the reader is referred to a paper by Langley and Anderson.¹

On the other hand, anatomists have lagged behind, the descriptions given in most text-books leaving the reader with no notion whatever of any ordered plan. The nomenclature of the anatomists is chaotic, and takes but little account of that used by physiologists.

It is scarcely to be wondered at that much confusion has arisen in medical circles. In one well-known work on neurology for instance, the sympathetic fibres to the bladder are depicted as leaving the spinal cord in the third, fourth, and fifth lumbar nerves, although in fact there are as a rule no white rami from these nerves. It would be easy to point out other errors of equal magnitude.

It is the purpose of the author to show that the autonomic nervous system in man is strikingly similar to that found in many animals, and that there is no need for a separate and dissimilar nomenclature. In this paper attention is confined largely to a consideration of the outflows to the lower abdominal and pelvic viscera. The sketch is incomplete, no attempt being made to review the literature exhaustively. The author endeavours rather to stress

outstanding facts which he has had an opportunity of verifying personally, and to present a clearly-defined picture which he hopes may be of service to medical men and to surgeons in particular.

PLAN OF THE AUTONOMIC NERVOUS SYSTEM.

It is assumed that the reader has a working knowledge of the plan of the autonomic nervous system as depicted by Gaskell and Langley. A description of the various outflows and arcs is beyond the scope of this paper.

NOMENCLATURE.

It is proposed to use largely the nomenclature employed by Langley. The nerve bundles passing forwards from the lumbar sympathetic trunks to the region of origin of the inferior mesenteric ganglion or ganglia will be called the 'inferior' or 'lumbar splanchnic nerves'. Certain bundles connecting the inferior mesenteric with the cœliac or superior mesenteric ganglion are named by Langley 'the ascending branch of the inferior mesenteric ganglion'. This appears to be an unsatisfactory title, as it is rather a root of the ganglion, and so it will be termed the 'cœliac root of the inferior mesenteric ganglion'. From the inferior mesenteric ganglion arise colonic branches, and the hypogastric nerves, right and left, which pass into the pelvis to take part in the formation of the pelvic plexuses. From certain sacral nerves arise the pelvic nerves which, after a short course, enter the pelvic plexus of the corresponding side. From the pelvic plexuses arise visceral branches to the rectum, bladder, and other pelvic organs. (*See Figs. 407, 408, 410, 411.*)

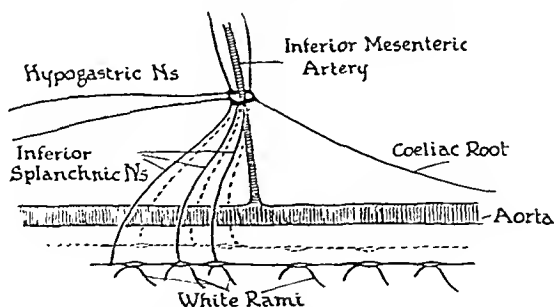


FIG. 407.—Diagrammatic representation of the lumbar outflow in the guinea-pig. The trunks lie close together. The lowest white rami enter the trunks well below the point of origin of the inferior mesenteric artery from the aorta. Similarly in relation to the artery the inferior splanchnic nerves arise low down and run ventrally and cranially on either side of the aorta to the inferior mesenteric ring, or ganglion, which is penetrated by the inferior mesenteric artery, as in other animals. The cœliac root enters the ganglion from above, and from the latter arise colonic branches and the hypogastric nerves. The trunks and inferior splanchnic nerves of the right side are represented by the interrupted lines. Actually the colonic branches are more numerous than shown in the diagram. Microscopically small ganglionic nodes are to be seen scattered in the 'ring'.

It is to be hoped that this nomenclature will displace the numerous terms found in anatomical text-books, such as 'aortic plexus', 'hypogastric plexus', 'nervus erigens', 'presacral nerve', 'intermesenteric plexus', and so on.

ANATOMY.

The macroscopic and microscopic anatomy of the autonomic nerves of the lumbar and sacral regions in rabbits, cats, and dogs was described in great detail by Langley and Anderson^{1,3} in a series of masterly papers. The author has made many dissections in dogs, cats, guinea-pigs, rabbits, opossums, monkeys, and human beings, and in all the general plan has been much the same, although minor variations are usually to be found.

The composition of the various nerve bundles has been studied by the method of teasing short segments in glycerin after immersion in a 1 per cent solution of osmic acid. In many instances nerves were divided in living animals and after the passage of six or seven days the animals were killed

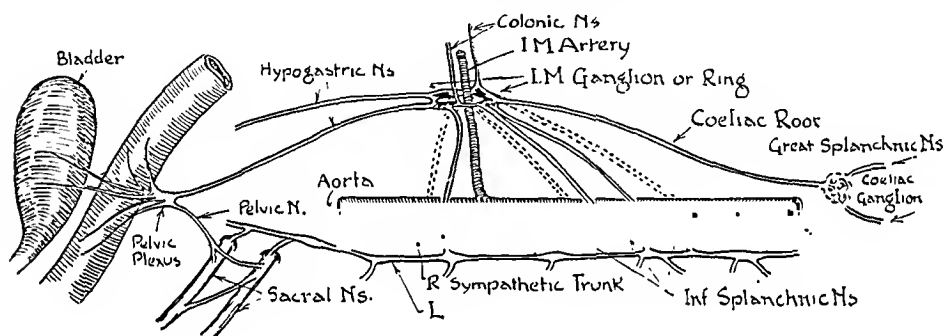


FIG. 408.—Diagram to show the arrangement of the lumbosacral nerves in the cat. The lumbar trunks lie close together. The inferior splanchnic nerves pass forward on either side of the aorta into the mesentery of the colon. In the region of the inferior mesenteric artery they unite to form stout bundles on either side united by decussating bands passing cranially and caudally to the artery so as to form a ring of nervous tissue encircling the vessel. The coeliac root passes into the ring, and from the latter arise numerous colonic branches and the hypogastric nerves. Several small ganglionic nodes are to be demonstrated in the 'ring', which, however, appears on superficial examination to be a large discrete nodule (see Fig. 414). The hypogastric nerves join up with the pelvic nerves to form the pelvic plexuses, which give branches to the pelvic viscera. The lesser splanchnic and renal nerves are not shown.

and degenerated fibres were traced. No attempt was made to count the fibres or to assess accurately the relative numbers of medullated and non-medullated fibres.

The Sympathetic Trunks (Figs. 408, 410, 411).—In the thoracic region each sympathetic trunk crosses the heads of the ribs and thus is situated quite close to the corresponding intercostal nerves. In the lower thoracic and lumbar regions the psoas muscle intervenes and the trunk passes forwards, skirting the medial border of the muscle and coming to lie on the antero-lateral aspect of the vertebral bodies a considerable distance from the lumbar nerves. Below the pelvic brim, the sacral trunk comes to lie close to the sacral nerves as they issue from the sacral foramina, and ends by joining with its fellow of the opposite side in the ganglion impar, situated low down in front of the coccyx. It should be noted that in small animals the two lumbosacral trunks are separated by a very small interval, whilst in man the interval is disproportionately greater. In the lumbar and sacral regions the trunks are connected by numerous fine twigs. Ganglia are found to correspond in

position more or less regularly with the vertebral bodies, but this is not an invariable arrangement. Fusion of two ganglia is frequently to be noted. The trunks between obvious ganglia contain both medullated and non-medullated fibres, the latter preponderating. The medullated fibres tend to remain near the surface and run in bundles (Müller²). Between the fibres are numerous scattered ganglion cells.

The Rami.—In the thoracic region the rami are quite short and the grey and white lie close together. In the lumbar area the grey rami pass almost directly backwards from the lateral chain to the appropriate spinal nerve accompanying the lumbar vessels under the arches of origin of the psoas muscle. They anchor the ganglionated trunks firmly so that the latter cannot be drawn ventrally until the grey rami are divided. Only a very short segment of a lumbar grey ramus is visible until the psoas muscle is removed (*Fig. 409*). The white rami take a long oblique course between the slips of

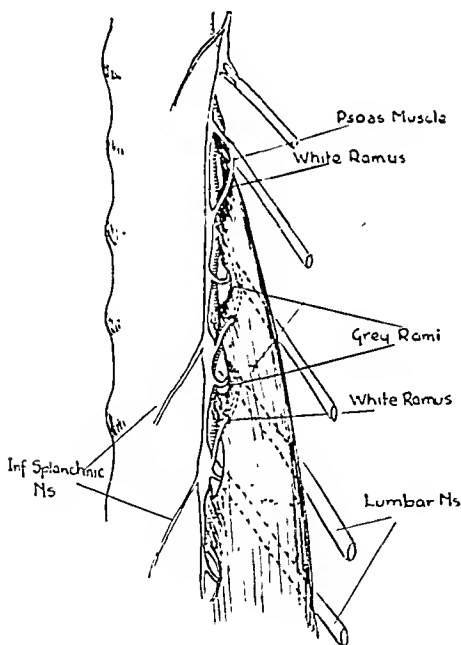


FIG. 409.—Diagram to show how the psoas muscle separates the lumbar sympathetic trunk from the spinal nerves. In the thorax, cranial to the highest slip of origin of the muscle, the trunk crosses the rib heads and lies quite close to the intercostal nerves, so that the rami are short and close together. In the lumbar region the trunk lies some distance from the lumbar nerves, so that the rami take a much longer course. The grey rami pass almost directly backwards, or backwards and downwards, from the trunk to the lumbar nerves in company with the lumbar vessels under the arches formed by the psoas muscle between the intervertebral discs. The white rami are obliquely placed, crossing the slips of origin of the psoas muscle and thus issuing from the surface of the muscle to join the trunks well below their point of origin. The grey rami always hug the vertebral bodies and never pierce the surface of the psoas muscle.

origin of the psoas muscle downwards, inwards, and forwards, to join the lumbar trunk well below their points of origin from the spinal nerves. They thus emerge from the ventral surface of the psoas muscle and are quite distinct from the grey rami. The white ramus from, say, the first lumbar nerve joins the trunk at or below the point of origin of the grey ramus in the second lumbar nerve (*Figs. 409-411*). The white rami are frequently duplicated and there may be more than two bundles (*Fig. 410*). In the dog the lowest white ramus springs from the third or fourth lumbar nerve, in Macaque monkeys usually from the third, though occasionally from the fourth, and in man from the second lumbar nerve. In the sacral region the grey rami are short and

pass obliquely outwards and downwards to join the sacral nerves. White rami do not arise from the nerves taking a major part in the formation of the limb plexuses.

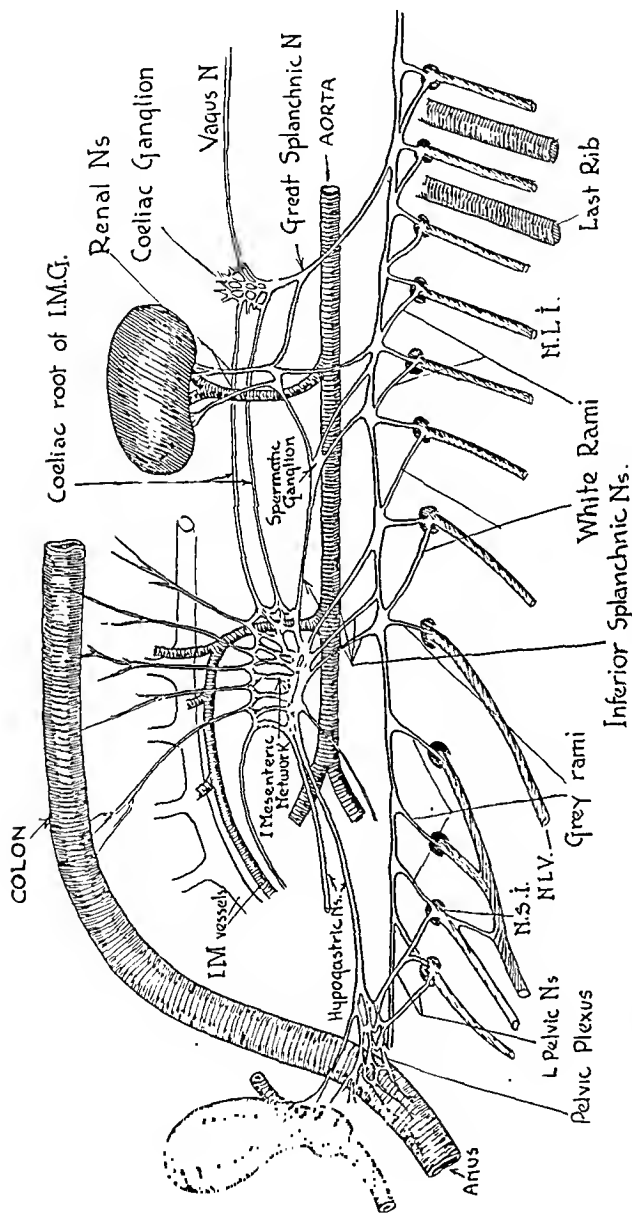


FIG. 410.—Diagram to show the arrangement of the lumbosacral autonomic nerves in Macaque monkeys, as seen from the left side, the kidney being shown ventrally. In the lumbar region the trunks are separated from the spinal nerves by the psoas muscle (not shown), so that the rami take a long course, the white passing obliquely between the slips of origin of the muscle and issuing from its ventral surface, the grey passing more directly and in company with the lumbar vessels under the arches formed by the muscle between its points of origin from the intervertebral discs. The coeliac roots join up with the uppermost inferior splanchnic nerves in a ganglionated network penetrated by the inferior mesenteric artery. From this network branches pass to join the thick paired ganglionated bundles formed by the union of the other inferior splanchnic nerves of each side. These bundles are connected by a stout decussating strand at their caudal extremities. Colonic branches spring from this network and bundles, and the hypogastric nerves arise from the caudal extremities of the lateral bundles. These nerves give branches to the ureters and pass on to the pelvis to take part in the formation of the pelvic plexuses. The left pelvic nerve is double in this case. The grey rami in the sacral region should have been drawn shorter, the trunks lying quite close to the sacral foramina. The inferior mesenteric ganglia are multiple and are found scattered in the network about the artery and in the paired lateral bundles below this level.

The white rami consist very largely of medullated fibres, which vary greatly in size. The grey rami are composed mainly of non-medullated fibres, but medullated fibres are commonly to be seen in fair numbers. The

proportion of medullated fibres appears to vary a good deal in different animals, but the author has not examined sufficient material to be worthy of record. Langley¹ discusses the question of origin and destination of the medullated fibres found in grey rami. They may be preganglionic fibres which have not made connection with cells in the ganglia of the lateral chains, but are passing to do so with cells situated further afield. They may be medullated postganglionic fibres, while some may be afferent fibres passing from the periphery via the grey rami to the trunks and so via the white rami to the appropriate posterior root ganglion.

The Lesser Splanchnic Nerves (*Figs. 410, 411*).—Below the lowest root of the great splanchnic nerve, arising from the thoracic and the upper lumbar trunks, are a varying number of branches which pass to the region of origin of the renal vessels. These freely anastomose and give off branches to the kidneys and adrenal glands, some to the celiac ganglion, and often one or more branches to the region of the inferior mesenteric ganglion or network and the spermatie or ovarian ganglion. These nerves are composed mainly of medullated fibres. Whilst they are described as branches arising from the trunks, probably the great majority of the fibres arise from cells in the spinal cord and merely pass through the trunks without making contact with ganglion cells situated therein.

The Inferior or Lumbar Splanchnic Nerves (*Figs. 407, 408, 410, 411*).—This name should be reserved for nerves passing to the inferior mesenteric ganglion and should not include the lesser splanchnic nerves arising from the upper lumbar trunks. They arise from the upper lumbar trunks and pass medially downwards and forwards round the aorta to reach the inferior mesenteric ganglion. On the right side they usually pass between the inferior vena cava and the aorta. They are variable in number (from two to four on each side) and point of origin, most commonly arising opposite the first, second, and third lumbar vertebræ. In man the more cranially situated of these nerves pass more directly forwards round the aorta and then turn sharply downwards in company with the celiac roots of the inferior mesenteric ganglion, often communicating with the spermatie or ovarian ganglion (*Fig. 411*). Hence, they have been described in anatomical text-books as branches 'reinforcing' the 'aortic plexus'.

The lesser splanchnic nerves and the inferior splanchnic nerves are composed in the main of medullated fibres of varying size. The fibres degenerate on the distal side of a point of section, or after section of the white rami, showing that the cell bodies are situated centrally.

The Celiac Root of the Inferior Mesenteric Ganglion (*Figs. 407, 408, 410, 411*).—This is constantly present and is usually represented by two or more nerve bundles springing from the celiac or superior mesenteric ganglion or from nerves in the vicinity. Frequently the lesser splanchnic nerves or renal nerves may add a contribution. The bundles pass downwards in front of the aorta, usually communicating with the spermatie ganglia, to join up with the inferior splanchnic nerves at the inferior mesenteric ganglion. Nerve fibres of the celiac root are mainly non-medullated, but medullated fibres are present in fair numbers. In dogs these degenerate on the caudal side of a point of section through the root at any point or after section of the great

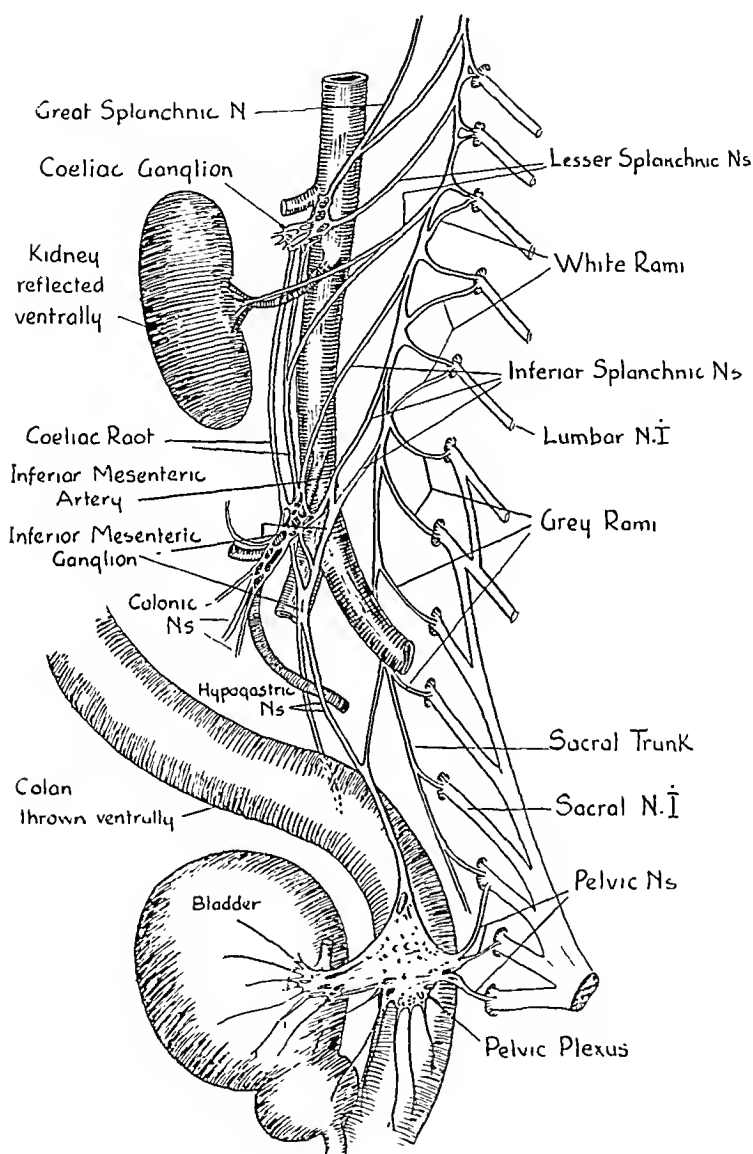


FIG. 411.—Diagram to show the arrangement of the lumbar and sacral autonomic nerves in man. The kidney and colon are thrown ventrally. The rami are much the same as in animals, and have the same relationship to the psoas muscle (not shown, see Fig. 409). The inferior splanchnic nerves and the coeliac root of the inferior mesenteric ganglion are clearly to be seen. The inferior mesenteric ganglion is similar to that shown in Fig. 112. The hypogastric nerves pass downwards into the pelvic plexuses. On the left side the hypogastric nerve receives a twig from the trunk. The pelvic nerve is represented by twigs from the second, third, and fourth sacral nerves. From the widespread ganglionated pelvic plexus numerous visceral nerves pass to the bladder, prostate, rectum, etc.

splanchnic nerves. Thus the nerve cells are probably situated in the thoracic portion of the spinal cord or in the posterior root ganglia.

The celiac root of the inferior mesenteric ganglion and the inferior splanchnic nerves have been described by Learmonth⁵ in recently published papers as the roots of the 'presacral' nerve. This author deplores the "too slavish a transference of the arrangement in animals to conceptions of human physiology". The present author is definitely of the opinion that there is a very close resemblance between the systems found in animals and man. Learmonth's terminology serves only to obscure the plan in man, and his description of the inferior splanchnic nerves and the inferior mesenteric network is distinctly misleading.

The Inferior Mesenteric Ganglion or Ganglia.—Langley and Anderson¹ point out that in the cat the ganglion consists of several distinct nodes, and they mention that Navrocki and Scabitchewsky (1891) had previously noted this fact. Many present-day authors appear to have overlooked this work.

In dogs, guinea-pigs, cats, and rabbits (*Figs. 407, 408, 413*) the ganglion is represented by several nodules in close proximity, connected by nerve bundles so as to form a ring of nervous tissue encircling the inferior mesenteric artery. The ring is usually made up as follows: Two or more of the inferior splanchnic nerves on each side fuse in a small ganglionic node which receives also twigs from the celiac root. A nerve bundle passing transversely cranial to the inferior mesenteric artery connects the two nodes, which may appear to be fused into a single mass, and fasciculi pass caudally from them on either side of the artery to enter small ganglia, which also receive the more caudally situated inferior splanchnic nerves. The lateral bundles are longer in rabbits than in cats and dogs (Langley and Anderson¹). The distal ganglia are connected by a short transverse nerve placed caudal to the artery, and give origin to the hypogastric nerves. From the ring of nerve tissue arise numerous nerves which run in company with the branches of the artery to the colon.

In Macaque monkeys (*Fig. 410*) the arrangement is very similar, but somewhat more complex. The celiac roots and the uppermost inferior splanchnic nerves of each side, which usually communicate with the spermatic or ovarian ganglion, enter a ganglionated network which is penetrated by the inferior mesenteric artery. From this plexus numerous colonic nerves arise, the uppermost of which may be traced to the region of the superior mesenteric plexus with which they appear to communicate, and also bundles passing caudally on each side of the artery, which are joined by the remaining inferior splanchnic nerves. Thus augmented, the thick lateral bundles run parallel for about 2 cm. At their caudal extremities they are firmly connected by a short stout transverse strand, and from this point the two hypogastric nerves arise and diverge. From the ventral aspect of the lateral bundles a large number of colonic nerves arise, which pass in the mesentery to the colon. Ganglionic nodes are to be found in the network which surrounds the artery and in the lateral bundles.

As the whole of the network lies between the peritoneal layers of the mesocolon, it is flattened in a sagittal plane and the lateral bundles at first sight appear to be one thick bundle (*Fig. 410*).

In man the arrangement is again very similar (*Figs. 411, 412*). The coeliac root, represented by several small nerves as a rule, passes into a network of nervous tissue which is penetrated by the inferior mesenteric artery. Into this network pass also stout contributions from the more cranially situated of the inferior splanchnic nerves. The latter give origin in addition to fasciculi which join up with the more caudally situated inferior splanchnic nerves to form thick ganglionated nerve bundles on either side of the mid-line. These

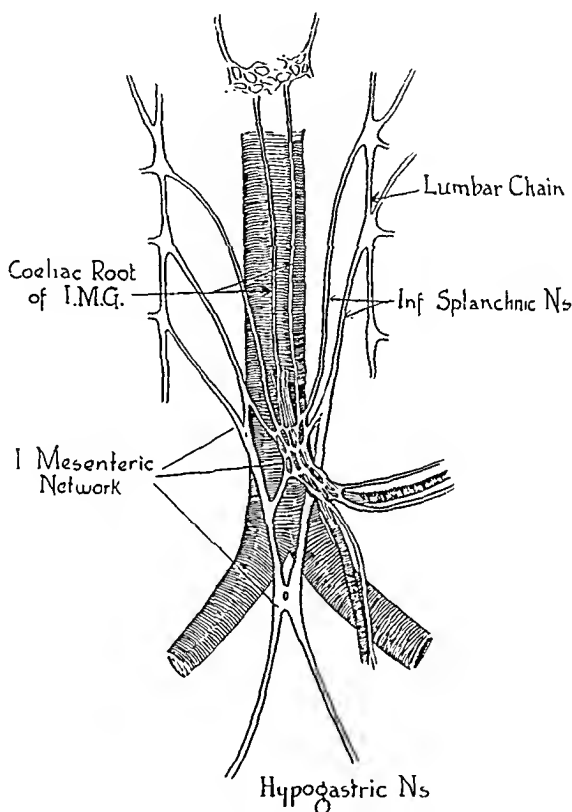


FIG. 412.—Diagram to show the formation and disposition of the inferior mesenteric network or ring in man, which corresponds to the 'ganglion' of animals. The 'ring' is formed above by the meeting of the coeliac root and the inferior splanchnic nerves, and ends below at the point of divergence of the hypogastric nerves. The inferior mesenteric artery penetrates the ring. Ganglia are invariably to be found scattered throughout the network, fairly large nodes being present as a rule in the thick lateral bundles formed by the fusion of the inferior splanchnic nerves.

bundles converge and meet at a point usually some little distance below the bifurcation of the aorta, but occasionally higher up in the region of the proximal part of the inferior mesenteric artery. At the point of meeting of the bundles there appears to be a free decussation of fibres, and from this point also arise the hypogastric nerves, right and left. Small connecting twigs commonly pass between the various parts of the network just described.

Ganglionic nodes are to be found commonly in the bundles formed by the union of the inferior splanchnic nerves on each side, scattered throughout the network surrounding the inferior mesenteric artery, and at the point of union of the bundles at the lower end of the network. Seldom do the nodes reach very large proportions, but almost invariably there is a considerable amount of ganglionic tissue present, distributed as outlined above. Occasionally the network is compressed and then larger ganglia are to be seen.

For the reasons just stated it is difficult to define the inferior mesenteric ganglion in man.

The whole of the network, extending from the points of union of the inferior splanchnic nerves on each side down to the point of emergence of the hypogastric nerves, and including the ganglionated network on the inferior mesenteric artery above described, is to be looked upon as representing in man the ganglionated ring seen in animals and known as the inferior mesenteric ganglion or ganglia. It has been pointed out that even in small animals where this appears to be a solitary node, it is not so in fact. In animals the descending colon is provided with a free mesentery and the sympathetic trunks lie close together, so that the inferior splanchnic nerves of each side run towards the inferior mesenteric ganglion in close proximity. In addition the inferior mesenteric artery leaves the aorta almost at a right angle. Thus it comes about that the inferior mesenteric ganglionated network is compressed and appears at first glance to be a discrete ganglion. In man the trunks are widely separated, the inferior splanchnic nerves converging sharply towards the mid-line. There is no free mesentery and the inferior mesenteric artery lies practically on the ventral wall of the aorta, which effectively prevents any compression of the inferior mesenteric ganglionated network. These factors probably explain why the network spreads laterally so much more in man than in animals.

A study of several microscopic sections of a fetus (about four months) showed clearly the multiple nature and bilateral arrangement of the ganglia in the region of the inferior mesenteric artery, although in this case they were situated close together as in small animals (Fig. 414).

Learmonth⁵ remarks that in man there is no ganglion corresponding to the inferior mesenteric ganglion of animals, or that it is small and of little importance. He ignores the fact that in animals the ganglion is in the nature

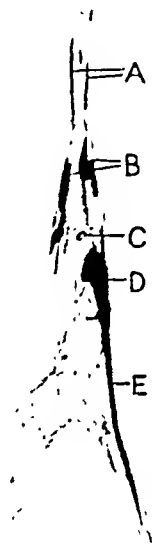


FIG. 413.—Photograph of a coronal section through the inferior mesenteric ganglion of a cat. The various nerves were stretched out before embedding in paraffin in order to ensure that they would lie in the same plane. Above, the inferior splanchnic nerves (A) and the colic root enter the 'ring'. Paired proximal ganglia (B) are plainly seen. Lateral bundles pass from these on either side of the inferior mesenteric artery (C) into ganglia (D) situated caudal to the vessel. One of these is not seen in the section. An indication of the transverse bundle connecting the two distal ganglia is seen. The hypogastric nerves (E) diverge below. ($\times 3$.)

of a ganglionated network, and that the same network in man contains a considerable amount of ganglionic tissue. Coates⁶ refers to 'rudimentary ganglia'. Why it should be assumed that the ganglia are rudimentary (Coates) or of little importance (Learmonth) is not clear. Leriche and Fontaine⁷ pointed out that there are scattered cells and microscopic ganglia in the 'presacral nerve' amongst others. They consider that their 'discovery' of these is enough to invalidate certain conclusions arrived at by Langley after experiments in which nicotine was applied locally to the inferior mesenteric ganglia. It was, of course, well known to Langley that many of the fibres

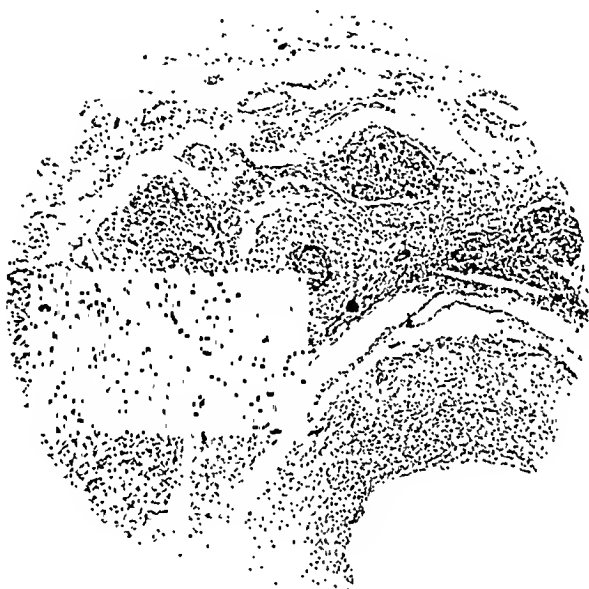


FIG. 414.—Photomicrograph of a horizontal section through the region of the inferior mesenteric ganglion in a foetus (about four months). Ventral to the aorta is seen the inferior mesenteric artery surrounded by ganglia and nerves. Serial sections showed distinctly the bilateral nature of this 'ring' and the multiplicity of ganglia. ($\times 46$.)

of the inferior splanchnic nerves pass by the inferior mesenteric ganglion to relay at cell stations further afield, and he did not neglect this fact in arriving at his conclusions.

The Hypogastric Nerves.—In animals these are represented by two or more nerves which arise from the inferior mesenteric ganglion and, diverging, pass downwards, not in company with the inferior mesenteric artery, to take part in the formation of the pelvic plexuses. There are frequently small communicating strands uniting the hypogastric nerves with the lumbosacral sympathetic trunks, which are to be looked upon as subsidiary inferior splanchnic nerves. In man the arrangement is much the same. The inferior mesenteric ganglionated network divides below into two complex fasciculated bundles, one passing on either side of the rectum to enter the corresponding pelvic plexus. The hypogastric nerve bundles contain scattered nerve cells

and sometimes small ganglionic nodes. These are to be regarded as outlying parts of the inferior mesenteric ganglion. As in animals, the hypogastric nerves frequently receive reinforcing twigs from the lumbar or sacral trunks, which may pass posterior to the external iliac arteries. They give several branches at different levels to the ureter of the corresponding side. The hypogastric nerves are composed of large numbers of non-medullated and many medullated fibres. The majority of the medullated fibres degenerate after section of the inferior splanchnic nerves, hence the nerve cells are situated in the lumbar segments of the spinal cord. The fibres decussate in part in the inferior mesenteric ganglion, so that each hypogastric nerve receives fibres from the inferior splanchnic nerves of each side of the body (Langley and Anderson⁸). Langley and Anderson¹ point out that after degenerative section of one pelvic nerve some of the fibres of the corresponding hypogastric nerve degenerate. In the cat these are very few, in the rabbit they are more numerous. The author has noted that in monkeys a few fibres of the hypogastric nerve degenerate after section of the pelvic nerve of the same side. The destination of these fibres has not yet been traced. No similar observations in man are as yet to hand.

The Pelvic Nerves (*Figs 408, 410, 411*).—Fine twigs, arising from two or more of the sacral nerves just after they issue from the sacral foramina, unite to form the pelvic nerves, one on either side of the body. Occasionally the nerve may be represented by two bundles. In dogs, cats, and monkeys the pelvic nerve is usually single, discrete, and about an inch or more in length. It passes distally into the pelvic plexus. In man the nerve twigs pass almost at once into the pelvic plexus, so that, as a rule, no definite single nerve may be demonstrated. The pelvic nerves are composed almost entirely of medullated fibres of varying size.

The nerves arise as a rule from two or more of the sacral nerves, but tend to vary somewhat, as do other plexuses, in actual level. Thus in the cat the main outflow is from the second sacral nerve, with variable contributions from the first and third (Langley). In the dog the second and third sacral nerves supply the main contributions. In Macaque monkeys the pelvic nerves arise from the first and second sacral nerves, in man from the third and either the second or the fourth, or both.

The Pelvic Plexus (*Figs. 408, 410, 411*).—This is a network composed of interlacing twigs resulting from the breaking up of the hypogastric and pelvic nerves. In it are situated numerous small ganglionic nodes. The network spreads out in a fan-shape from the points of entry of the above-named nerves, which are fairly close together. From the plexus many fine twigs radiate to the different organs in the pelvis, most of which are mixed nerves containing fibres from both of the parent nerves or from the ganglia of the plexus. In man the network is well developed and its branches are very numerous. Ganglionic nodes are more numerous near the point of entry of the hypogastric nerve. The visceral branches of the pelvic plexuses are composed of non-medullated and medullated nerve fibres. The former probably belong mainly to the lumbar sympathetic outflow, and have their cells situated either in the ganglia of the inferior mesenteric network or the pelvic plexus. The latter belong almost entirely to the sacral outflow, as they degenerate after

section of the pelvic nerves (dogs and monkeys). The cell stations for this group of fibres are situated on or in the walls of the viscera in question, at least in part.

REFERENCES.

- ¹ LANGLEY, J. N., and ANDERSON, H. K., "The Innervation of the Pelvic and Adjoining Viscera", *Jour. of Physiol.*, 1896, xx, 372.
- ² MÜLLER, L. R., *Die Lebensnerven*, 1924, 16.
- ³ LANGLEY, J. N., and ANDERSON, H. K., "On the Innervation of the Pelvic and Adjoining Viscera", *Jour. of Physiol.*, 1895, xviii, 62; xix, 71, 85, 122, 131; 1896, xix, 372; xx, 372.
- ⁴ LANGLEY, J. N., "The Sympathetic and Other Related Systems of Nerves", *Schafer's Text-book of Physiology*, 1900, ii, 616.
- ⁵ LEARMONTH, J. R., "A Contribution to the Neurophysiology of the Urinary Bladder in Man", *Brain*, 1931, liv, Pt. 2, 147.
- ⁶ COATES, A. E., "A Note on the Macroscopic Anatomy of the Nerves of the Bladder", *Med. Jour. of Australia*, 1932, ii, 683.
- ⁷ LERICHE, R., and FONTAINE, R., "Quelques Faits nouveaux touchant l'Anatomic normale du Sympathique, basés sur l'Examen histologique de 40 Pièces opératoires", *Presse m'ed.*, 1929, xxxvii, 903.
- ⁸ LANGLEY, J. N., and ANDERSON, H. K., "The Constituents of the Hypogastric Nerves", *Jour. of Physiol.*, 1894, xvii, 177.

MALIGNANT ADENOMA OF THE PROSTATE WITH SECONDARY GROWTHS IN THE VERTEBRAL COLUMN SIMULATING POTT'S DISEASE.

By W. E. CARNEGIE DICKSON,

PATHOLOGIST, WEST END HOSPITAL FOR NERVOUS DISEASES, LONDON,

AND T. R. HILL,

ASSISTANT PHYSICIAN AND PATHOLOGICAL REGISTRAR, WEST END HOSPITAL FOR NERVOUS DISEASES, LONDON.

THIS case is described for two reasons. In the first place, the clinical and pathological findings in the central nervous system were of such interest as to deserve being placed on record. Secondly, the case was a good example of the well-recognized but often extremely confusing picture that results from the remote metastases of a hidden primary focus of malignant disease. In such a case as the present one, the youth of the patient adds to the diagnostic difficulties by tending to divert suspicion from the possibility of such a hidden primary focus and also from its particular site—in this instance the prostate gland.

HISTORY.—The patient was a man aged 30, a printer, who was well until four months before admission to the West End Hospital for Nervous Diseases. His first symptoms were pain and stiffness low down in his back, appearing insidiously, and the condition had been diagnosed as “museular rheumatism”. The symptoms grew gradually more severe, and the next development was that the patient felt as though he were “walking on rubber”, and weakness of all four limbs slowly appeared and progressed. Three weeks before admission the patient was quite unable to stand and found it difficult to pass urine. Numbness had spread to the abdominal wall. From this time onwards the condition progressed rapidly until spastic paraplegia with distension-overflow from the bladder and retention of fæces were marked, with complete anæsthesia of trunk and legs below the nipple line and with great weakness of the hands. The intrinsic muscles of both hands showed wasting and other signs of lower motor neuron atrophy. An ascending urinary infection had developed, the urine being alkaline and containing much pus and albumin and enormous numbers of mixed organisms, e.g., streptococci and staphylococci, gram-negative and gram-positive bacilli; *Bacillus proteus* predominated in the cultures.

ON EXAMINATION.—A marked chronic pustular acneiform eruption was present over the back, and the pustules contained *B. proteus*—presumably secondary to the bladder infection—in addition to *Staphylococcus albus*. An examination of the cerebrospinal fluid obtained by lumbar puncture showed a definite Froin's syndrome—xanthochromia and fibrin clot on standing—with approximately 1 per cent of total protein and an extremely marked

excess of globulin. Cell-count: 5 small lymphocyte-like cells per c.mm. in the first fraction drawn off; in the second, 3 cells per c.mm. Wassermann and Kahn reactions negative (these being also negative in the blood). Lange's colloidal gold curve 0001223332.

A cisternal injection of lipiodol was given and the patient was X-rayed, cerebrospinal fluid from the cistern being examined. The normal findings in this may be compared with those of the lumbar puncture fluid described above. It was clear and colourless. Cell-count: 2 small lymphocyte-like cells per c.mm. Total protein: 0.02 per cent. Globulin: no excess. Wassermann and Kahn reactions negative, and Lange curve 0011000000.

A blood-count showed some degree of polymorphonuclear leucocytosis: total leucocytes 15,200 per c.mm., polymorphs 80 per cent.

The X-ray findings are described below. A diagnosis of a spinal tumour in the lower cervical region was made. Death occurred suddenly following an attack of vomiting and before laminectomy could be carried out.

PATHOLOGICAL EXAMINATION.—Those parts of this examination which are relevant are described. The body was that of a well developed young adult male with a profuse pustular eruption over the back.

Removal of Spinal Cord.—On beginning this removal the spinous processes of the 7th cervical and 1st dorsal vertebrae were seen to be unusually prominent and they were distinctly movable. The laminae of these vertebrae, on sawing them through to open up the spinal canal, were found to be pathologically softened. When the spinal cord had been exposed the lower part of the cervical enlargement was found to be markedly softened also. The bodies of the 7th cervical and 1st dorsal vertebrae showed an abnormal degree of projection backwards, so that they had pressed on the cord, causing the softening and tending to occlude the spinal cord.

The spine at the level of the bodies of the two affected vertebrae was unduly movable, producing the general appearance of a fracture-dislocation supervening upon disease of the bone such as neoplasm or tuberculosis. The head and neck of the first rib on the right side also showed extensive softening.

A portion of the spinal column from the 6th cervical to the 3rd dorsal vertebra inclusive was removed with the proximal parts of the ribs attached, so as to include all the diseased area. An X-ray photograph was taken of it (*Fig. 415*), and the specimen was then divided sagittally (*Fig. 416*). The bodies of the 7th cervical and the 1st dorsal vertebrae are seen to be almost entirely destroyed by disease, and collapse has taken place, so that the corresponding intervertebral discs are practically touching one another. The middle one of these three discs has been displaced backwards, and, as shown in the illustration, has been the chief agent in compressing the spinal cord. The body of the 2nd dorsal vertebra shows considerable softening posteriorly, but remains uncollapsed.

The disease process had also spread laterally so that the transverse processes of the 7th cervical and 1st and 2nd dorsal vertebrae were invaded and softened, the right side being most affected. The articular surfaces of the 1st and 2nd ribs on the right side were involved and the head of the former partially destroyed. The lateral attachments of the 1st dorsal neural arch



FIG. 415.—Radiogram of specimen after removal. It includes from the 6th cervical to the 3rd dorsal vertebra, with the proximal portions of the upper three pairs of ribs. The neural arches have been cut away for the removal of the cord. The bodies of the 7th cervical and 1st dorsal vertebrae have collapsed, and the three intervertebral discs above, between, and below have been crushed together and are almost touching one another. There is also some rarefaction of the body of the 2nd dorsal vertebra, and of the transverse processes of the 1st and 2nd dorsal vertebrae on the right side, as well as of the heads and necks of the 1st and 2nd right ribs.

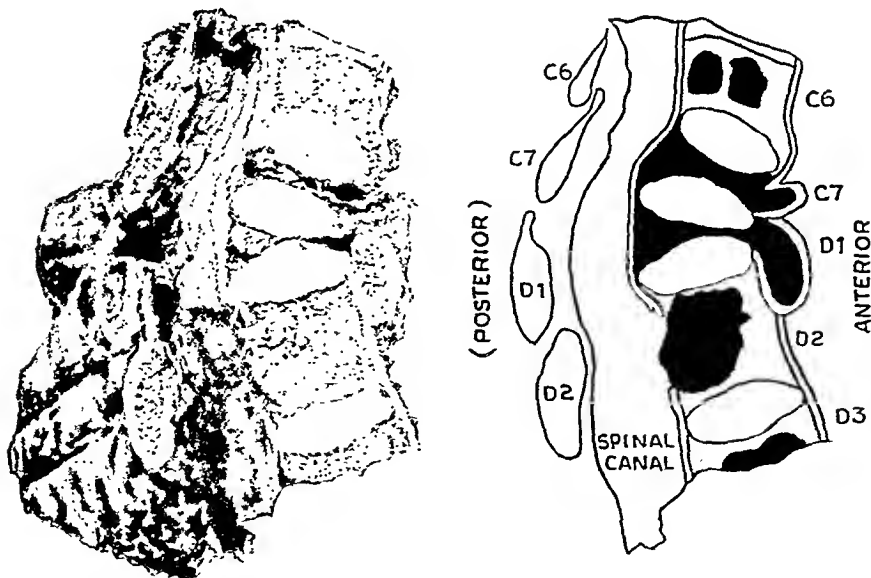


FIG. 416.—Mesial vertical section through specimen after removal, the corresponding radiogram of which is shown in Fig. 415. The collapsed condition of the bodies of the 7th cervical and 1st dorsal vertebrae is shown, and also the extensive malignant growth in the still uncollapsed body of the 2nd dorsal and slighter involvement of the 6th cervical and 3rd dorsal. The backward thrust of the three intervertebral discs, especially the middle one, into the spinal canal, by which the cord was compressed, is also shown. The areas invaded by the tumour are shaded in black in the key sketch. ($\times \frac{1}{2}$).

were affected, this leading to the movability of the spinous process as noted. The 1st dorsal nerve-roots on both sides were involved in the foramina and seemed totally destroyed.

The tissue in the softened areas of bone was of a pale yellowish-white colour, granular and crumbling. Much pseudocystic cavitation was present. The impression was gained that this tissue consisted of a metastatic deposit of a malignant tumour, and was not tuberculous.

Spinal Cord.—No evidence of neoplastic involvement of the cord itself was found. Naked-eye and microscopical sections of the compressed area showed generalized non-inflammatory degeneration, the white matter being fenestrated and spongy and the ganglion cells of the grey matter all showing advanced necrotic changes.

Thorax.—Well-marked hypostatic bronchopneumonia was present in both lungs, and the myocardium showed cloudy swelling and fatty changes.

The bronchial lymph-glands at the roots of the lungs were slightly enlarged, especially on the left side. They were, in parts, unusually pale and granular, and were removed for section, as were also some enlarged lateral inferior deep cervical (supraclavicular) glands.

Prostate.—This was only very slightly enlarged, but on section there was some slight suggestion of nodulation of the cut surface. Recent thrombosis of the veins of the prostatic plexus was present; and there was some enlargement of the neighbouring pelvic lymphatic glands, as well as of a few of the retroperitoneal glands along the course of the abdominal aorta.

Microscopical Examination.—Sections of the prostate showed the presence of an adenoma which had become malignant (*Figs. 417, 418*).

The enlarged lymphatic glands examined—pelvic, bronchial, and cervical—showed infiltration with the same malignant growth.

Figs. 419 and 420 show a low- and a high-power view of the tissue in the softened areas of the spinal column and reveal the presence of a metastasis of the same tumour. Very little bone is left.

SUMMARY.

A case of primary prostatic adenocarcinoma in a man of 30 is described. Metastases in the pelvic, prevertebral abdominal and thoracic, and deep cervical lymph-glands and in the bone of the vertebral column were found, the latter involving the bodies of especially the 7th cervical and 1st dorsal vertebrae and adjacent portions of the spinal column and ribs, producing collapse of the vertebral bodies and a condition simulating Pott's "angular curvature", with compression and softening of the spinal cord, Froin's syndrome with xanthochromia, etc., and the sudden death of the patient. The difficulties of diagnosis are explained and the clinical course and pathological findings are outlined. The changes in the spinal column were an indication of the tendency of a prostatic neoplasm to metastasize in bone, and were localized to the region described.

The case therefore differs from the unusual case described by Roberts,¹ where there was a continuous direct spread of the growth on the intraspinal surface of the dorsal wall of the spinal canal from the sacral to the cervical

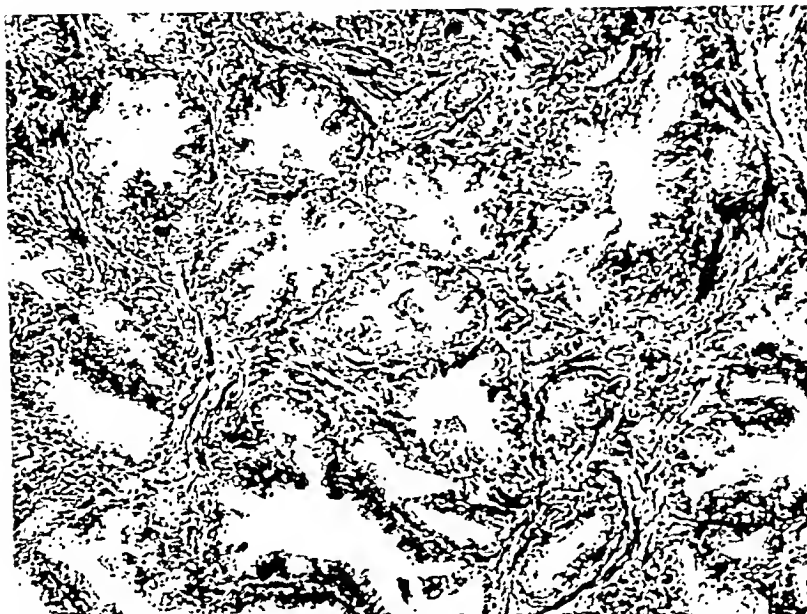


FIG. 417.—Low-power of the prostatic adenoma showing commencing malignancy. In other parts it has the appearance of a 'simple' adenoma. In this portion it is commencing to show increased complexity, and in other areas there is distinct malignant metaplasia of the epithelial cells and infiltration of the stroma. Compare with Fig. 418. (Van Gieson and hæmatoxylin.) ($\times 100$.)

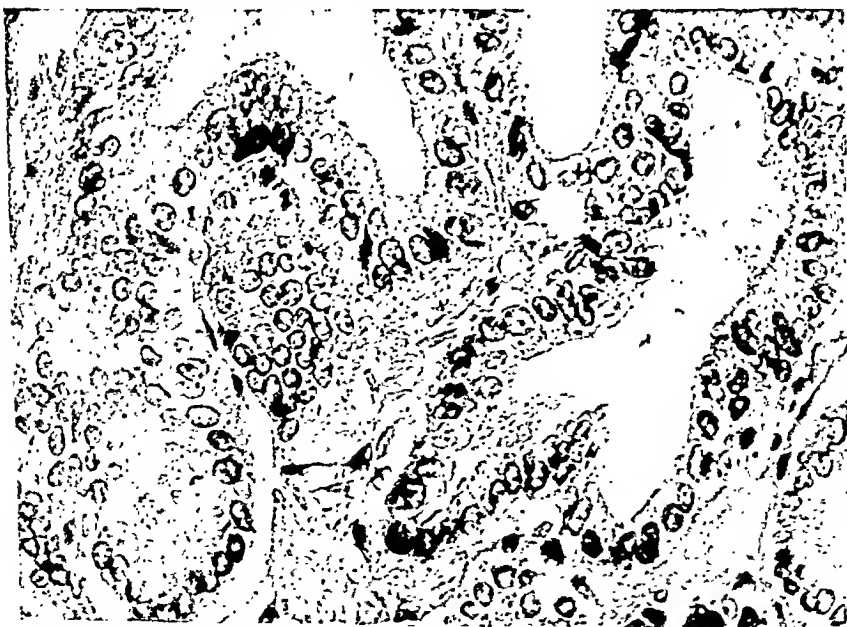


FIG. 418.—High-power of a portion of the prostatic tumour showing metaplasia to malignant adenoma or adenocarcinoma. There is active nuclear proliferation, and in some parts the cells form plasmoidal masses, with imperfect division of their cell-bodies. (Van Gieson and hæmatoxylin.) ($\times 400$.)



FIG. 419.—Low-power section of the tumour invading the body of the 1st dorsal vertebra. The tissue was cut without decalcification and is rather thick. It contained only a few small spicules of bone in process of absorption, and there was no irritative bony proliferation. (Hæmatoxylin and eosin.) ($\times 100$.)

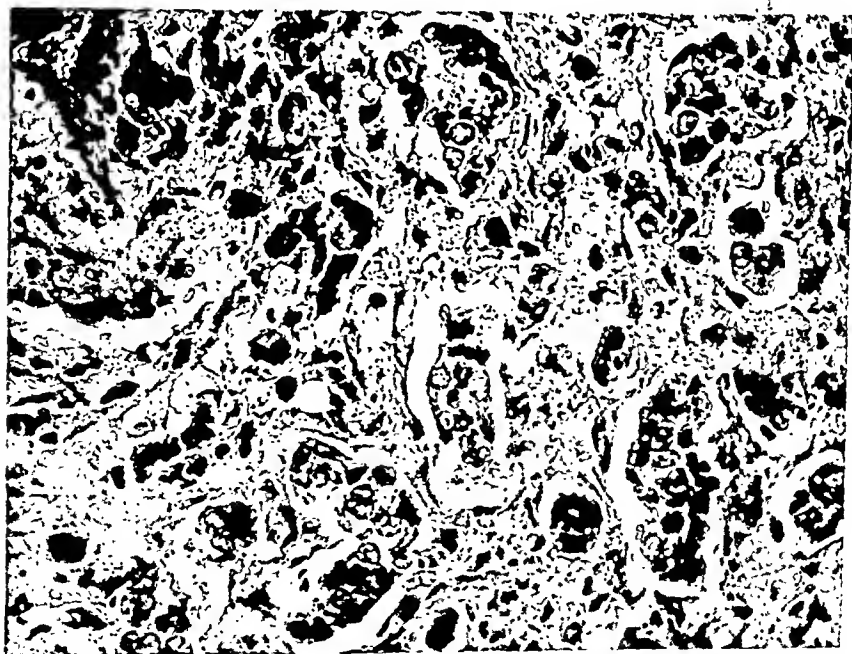


FIG. 420.—High-power of section seen in Fig. 419. A small fragment of partially absorbed bone is seen at the upper left corner. Numerous groups of the invading malignant glandular cells are shown. (Hæmatoxylin and eosin.) ($\times 400$.)

region, though his theory that there is an intraspinous pathway for the dissemination of prostatic carcinomata consisting of the spinal laminae with their ligaments and the lymph-spaces connected with these structures, might be considered in this case. It may also be mentioned that in the present case the bony metastases were completely osteoclastic and showed no evidence of the osteoplastic process which is generally described as characteristic of prostatic skeletal metastases.

We are indebted to Dr. E. D. Macnamara for the clinical notes of this case, which was under his care; to Dr. Martin Berry and his assistant Mr. A. York for the radiogram; and to Mr. J. Ottewill for his skilled technical assistance.

REFERENCE.

- ¹ ROBERTS, *Brit. Jour. Surg.*, 1928, xv, 652.

PRIMARY CARCINOMA OF THE LIVER:

WITH REPORT OF CASE SUCCESSFULLY TREATED BY PARTIAL HEPATECTOMY.

By A. LAWRENCE ABEL,

ASSISTANT SURGEON, CANCER HOSPITAL (FREE), LONDON.

CARCINOMA occurs only very rarely as a primary disease of the liver, in marked contrast to secondary growths, which are extremely common in this organ. Up to sixty years ago observers believed the reverse to be the case, and, in spite of Virchow's warning in regard to the importance of searching for a primary focus with meticulous care, many cases were reported which were later discredited.

HISTORICAL SURVEY.

Kelsch and Kiener²⁸ in 1876, in reporting two cases of primary carcinoma of the liver, were able to find only one other authentic case in the literature. Accurate knowledge of the subject really begins in 1884 with the work of Sabourin.³⁷ who added a series of four cases. Four years later Hanot and Gilbert²¹ presented a comprehensive contribution which marked a distinct epoch in the study of the disease. They described three gross types: (1) Nodular, (2) Massive, and (3) Cancer with cirrhosis; and recognized two varieties microscopically: (1) Alveolar; and (2) Trabecular. This remained the standard work for many years.

In 1901 Eggel¹³ collected 163 cases (of which only 117 had been studied microscopically) and added one of his own. He abandoned the idea of a separate cirrhotic type, because he believed that cirrhosis in some degree was present in all cases, and adopted the following classification from the point of view of gross morbid anatomy: (1) Nodular—where the liver contains several tumour nodules, varying in size, and generally well circumscribed; (2) Massive—where the tumour consists of one large mass, occasionally associated with metastases; (3) Diffuse—where the tumour process appears to extend throughout the whole liver substance.

Wegelin⁴⁶ in 1905 described 8 cases, but the next advance of note was made by Yamagiwa⁵² in 1911. He disagreed with the French writers in regard to the impracticability of their fine histological differentiation and made two simple divisions: (1) Hepatoma, i.e., carcinoma of hepatic cells; and (2) Cholangioma, i.e., carcinoma of bile-ducts. The term 'hepatoma' had previously been used by Sabourin in reference to a condition of nodular hyperplasia which in his opinion was a transitional stage between adenoma and carcinoma. Most modern writers, however, accept Yamagiwa's interpretation, and use it as a term for primary carcinoma of the liver cells. By 'cholangioma' is meant "only those carcinomata arising from the smallest bile canaliculi, and not those from other parts of the biliary apparatus".

Although later research work and literature have been voluminous, the broad views of Eggel, as modified by Yamagiwa, are now almost universally accepted as being the most satisfactory.

INCIDENCE.

There is no dearth of cases in the literature, and it is reasonable to assume that this is largely due to the general interest attached to a disease of such rarity. For this reason it is also probable that the majority of cases occurring during this century have been reported.

As secondary growths of the liver are so common and it is extremely difficult in all cases absolutely to exclude the possibility of a primary focus—such as, for instance, a small nodule in the kidney or adrenal—there is bound to be considerable overlapping in the available statistics from all sources. This would account to some extent for the wide variation that occurs in the reports of various observers. Added to this, there is no doubt that the incidence of the disease varies greatly in widely separated geographical areas.

An analysis of all available reports shows that the average incidence varies from 0.1 to 0.3 per cent of all autopsies in Europe and North America.

INCIDENCE OF PRIMARY CARCINOMA OF THE LIVER.

Winternitz ¹⁸	Analysis to 1916 Johns Hopkins Hospital	0.028–0.3 per cent 3 in 3700 cases = 0.08 per cent
Wheeler ¹⁷	Guy's Hospital to 1909	6 in 5000 cases = 0.1 per cent
Counseller and McIndoe ¹⁰			Analysis, Eggel to Mayo, 1926	62 in 42,276 cases = 0.08 per cent
Brines ⁶	Analysis, 1933	Average, 0.8 per cent
Clawson and Cabot ⁹	University of Minnesota, 1923	1 in 5100 cases = 0.002 per cent
Fried ¹⁶	Harvard, 1924	Only 1 reported case

Geographical Variations.—L. W. Smith⁴⁰ in 1926 found 12 out of 877 consecutive necropsies, i.e., 1.3 per cent, during one year in the Philippine Islands. Less than 70 of these deaths were due to malignancy, so that 17 per cent of all deaths associated with malignant disease were due to primary carcinoma of the liver. He mentions that “incidentally it is of passing interest to state that during the interval fully as many patients went home from the Philippine General Hospital with the clinical diagnosis of malignant disease of the liver as came to necropsy, and there seems to be no reason to doubt the clinical diagnosis as all the cases coming to necropsy were recognized ante-mortem.”

Strong and Pitts⁴¹ reviewed 9 cases collected at the Vancouver General Hospital (1920–7) out of a series of 1024 autopsies, giving an incidence of

0.87 per cent. Eight of these patients were Chinese, so that the percentage out of 115 autopsies on Chinese was 6.9 per cent. In the 909 autopsies on white patients primary carcinoma of the liver was found only once, i.e., 0.11 per cent, which agrees with the foregoing figures for white races.

Pirie³⁴ in 1921, writing at the South African Institute for Medical Research, concluded that carcinoma, generally speaking, is not so common in Africans as amongst Europeans, but in his experience primary carcinoma of the liver appears to be relatively more common.

If the figures are excluded for those where there is an abnormally high occurrence of the disease—due presumably to some local factor—the incidence does not exceed 0.8 per cent of all autopsies.

However, Brines⁶ recently gave a detailed report on 8 cases of primary carcinoma of the liver out of 1087 necropsies at Detroit Receiving Hospital (1929–32). This, with 3 additional cases referred to in brief, gives an autopsy incidence of 0.8 per cent, which is considerably higher than that usually recorded.

According to Jaffe,²⁵ primary carcinoma of the liver occurs in 1.5 to 3 per cent of all cancers.

Age Incidence.—Eggel, in his original survey, gave the average age incidence for men as 53.1 years, for women 52.2 years. Ewing¹⁴ stated that the disease occurs mainly between 40 and 60 years, especially between 50 and 60, while biliary carcinoma is relatively late, rarely occurring before the age of 40 years. However, a survey of the literature shows that no age is exempt. One patient in the Strong-Pitts series was 76 years old (average 45.6 years), while Müller³² recently reported a case in a patient who was 83 years of age.

At the other extreme of age, in the experience of numerous observers (Allbutt¹, Castle⁸, Dansie¹¹, Griffith¹⁹, Wollstein⁴⁹) the disease has been found in children of all ages and even in infants from the moment of birth. The occurrence of such early cases raises the possibility of some of these tumours being of an embryonal nature, and some at least of them have been described as such. It is agreed by these workers that though primary carcinoma of the liver does occur in infancy and childhood it is definitely rarer than in adult life, while the occurrence of sarcoma is of even greater rarity than carcinoma.

Sex Incidence.—Eggel's sex incidence figures were 63.3 per cent in men, 36.7 per cent in women; according to Karsner²⁶ also primary carcinoma of the liver occurs more frequently in men than women, and this is the universal opinion.

PATHOLOGY.

The liver is unique in its extraordinary powers of regeneration after trauma. In Mann's experiments on dogs³⁰ partial hepatectomy (four-fifths of the organ) was followed by complete regeneration in six weeks, and this reparative process was found to be accomplished by the hyperplasia of cells rather than by their hypertrophy. This remarkable capacity for regeneration in response to trauma and inflammatory conditions must always be considered in deciding on the malignancy, or potential malignancy, of a group of liver cells.

Local hyperplasia of liver cells may take several forms, and, as Ewing puts it, "all of these conditions occur with and without cirrhosis, and many of the simple hyperplastic processes lead through benign tumours into malignant neoplasms, thus complicating the task of separation into groups."

He makes three divisions of epithelial neoplasms of the liver: (1) Hepatoma; (2) Cholangioma; (3) Mixed tumours. A hepatoma is a solid parenchymatous tumour arising from liver cells; a cholangioma, which is the glandular type, arises from intrahepatic bile-ducts; while the third variety arises apparently from both liver cells and bile-ducts. From the point of view of gross anatomy they are "solitary or multiple, massive or nodular, and rarely diffuse". Microscopically he classifies hepatoma as: (a) Adenoma; (b) Adenocarcinoma; or (c) Carcinoma (solitary, multiple, or atypical). Cholangioma is classified as: (a) Adenoma (solid or cystic); (b) Adeno-carcinoma; or (c) Carcinoma.

Clinical Classification of Hepatoma (according to Ewing).—

a. Solitary adenoma, whose benign character is due chiefly to encapsulation.

b. Massive liver-cell carcinoma, which represents a more rapidly growing, atypical, and malignant form of the solitary adenoma (a).

c. Multiple liver-cell carcinoma, which includes the highly malignant, rapidly-growing tumours occurring in livers in which cirrhosis is either absent or slight.

d. Carcinomatous cirrhosis, where the tumour appears to be the direct sequel of, or essentially connected with, the cirrhosis.

Histogenesis.—Van Heukelom²³ was the first to show how "hypertrophic liver cords" developed into hepatoma, and this work was later elaborated by Goldzieher and Bokay.¹⁸ The transition of nodular hyperplasia into multiple adenoma and multiple carcinoma has also been studied^{23, 29}, and the process sometimes demonstrated in the same liver. In a similar manner the various gradations between adenomatous and carcinomatous cholangiomas have also been noted to follow proliferation of the intrahepatic bile-duct epithelium.

As regards the mixed type of tumour, the process is not so definitely established, and is still in doubt. Muir³¹ and Rolleston (quoted by Ewing) maintain that "typical hepatomas may produce canals resembling bile-ducts".

Sarcoma.—Primary tumours of mesoblastic origin are extraordinarily rare, and their exact histogenesis has never accurately been worked out. According to Ewing there is "somewhat scanty but direct evidence that both the stroma cells and the endothelium of the capillaries are susceptible to tumour growth". Many of the reported cases probably represent mistakes in diagnosis, possibly due to incomplete autopsies. Syphilitic lesions in infants (Rolleston¹), and leukaemia (Ewing), are both potential sources of error. Histologically they may be grouped as: (1) Angio-sarcoma; (2) Alveolar sarcoma; (3) Spindle-cell sarcoma; (4) Round-cell sarcoma.

Incidence.—According to Jaffe, "approximately 48 true primary sarcomas of the liver have been reported to date" (1924). Clinically, they cannot be distinguished from carcinoma, and run a parallel, though more rapid, course.

Primary Melanoma of the liver "is a very difficult diagnosis to establish" (Ewing). Rolleston, in reviewing the reports of 9 cases, emphasizes the possible relation with the uveal tract, and mentions that in at least two cases very small uveal melanomas were found post mortem.

Sarco-carcinoma.—The occurrence of this composite tumour, associated with cirrhosis, has been reported by both Lubarsch²⁹ and Saltykow.³⁸

Separate Formation of Sarcoma and Carcinoma in the Same Liver.—Jaffe refers to two cases observed by Dominici and Merle,¹² and Saltykow, respectively. To these he adds a full report of a third case of his own, occurring in association with cirrhosis of long duration.

Origin of Primary Carcinoma.—Opinion is fairly equally divided on the respective theories of the unicentric^{26, 35, 48} and multicentric^{10, 23, 43} origin of these tumours, and there is apparently no likelihood of an early decision on the question.

ETIOLOGY.

1. Cirrhosis of the Liver.—The importance of cirrhosis as a frequently associated lesion has been mentioned by almost every observer since the work of Sabourin. Yamagiwa gave its association with hepatoma as being 75 to 100 per cent, and for cholangioma 50 per cent.

While practically all writers concur on this point, there is no agreement on its rôle as an etiological factor. In this connection there are three possibilities: (a) The associated cirrhosis may be a coincidence; (b) It may follow the tumour formation; or (c) It may precede it. The frequency of its occurrence, noted so universally, prevents acceptance of the first theory. As regards the second, cases have been observed in which an advanced carcinoma was present in the absence of definite cirrhosis.²² On the other hand Blumenau⁴ in 1920, in an investigation on hepatic cirrhosis, found that primary carcinoma of the liver had occurred in 3.5 per cent of a series of 198 cases of death from cirrhosis. This relatively high incidence of primary carcinoma is an obvious argument in favour of the etiological influence of an antecedent cirrhosis.

Counsellor and McIndoe¹⁰ noted that the predominance of males in the sex incidence of primary carcinoma corresponds to that of cirrhosis, and concluded that "from 3 to 4 per cent of cases of cirrhosis, particularly of the portal type, will become carcinomatous". They believed also that cirrhosis is associated with almost all hepatomas, and though it does not appear to be so frequently observed in cholangiomas, this may be due to the insufficient number of recorded cases.

It is interesting to note that Fried¹⁶, in giving his opinion that hepatoma is more frequent in males, cholangioma in females, gave as a possible explanation "the predominance of cirrhosis of the liver in men, and bile-duct infections followed by an inflammatory hyperplasia of bile-ducts in women".

The consensus of opinion^{36, 47} appears to be in favour of cirrhosis being antecedent; a few writers consider it a sequel to the neoplastic process, while others believe that the cirrhosis and carcinoma are both end-results of a common stimulating factor.

2. Chronic Parasitic Infections.—The importance of chronic hepatic disease as a causal factor is emphasized by the relatively high incidence of primary carcinoma of the liver in other races that are prone to various parasitic infections.

Thus Pirie in South Africa (1921) refers to the marked frequency of schistosomiasis (i.e., hemic distomiasis) in his cases, and suggests this as a definite causal factor in producing carcinoma of the liver, since it has been experimentally shown to give rise to cirrhosis. In this connection Fairley¹⁵ (quoted by Fried) maintained that the associated cirrhosis is due not to the ova alone, but mainly "to the bilharzial toxin present in the blood-stream, which is filtered out by the liver".

Smith, working in the Philippine Islands in 1926, where primary carcinoma of the liver is so common as "a definite disease entity" that it can readily be diagnosed, stated that "the cirrhosis, as far as is known, invariably precedes the tumour formation, and is commonly conceded as being due to chronic infections of intestinal origin, either bacterial or parasitic".

Strong and Pitts in 1930, in their series of 9 cases, stated that 8 of these patients were Chinese who "came entirely from Kwantung, a southern province of China, where liver fluke (*Clonorchis sinensis*) is common, and where chronic diseases of the liver frequently occur" (hepatic distomiasis).

Jaffe quoted several authors who noted echinococcosis in association with the disease, while Ascoli³ found that all his cases gave a history of syphilis, malaria, or alcoholism.

With regard to the etiology of the cirrhosis itself, many factors have been mentioned apart from the various parasitic infections. These include alcohol and cardiac disorders, while Karsner includes coffee, tobacco, and opium as playing an important part in its production.

3. Etiological Factors of Minor Importance.—Amongst these may be mentioned a history of hereditary cancer (and this was present in 4 of Eggel's cases), lues, and trauma. As regards the latter, Hicks²⁴ in 1929 recorded a case of encapsulated carcinoma in a man of 77, who gave a definite history of severe trauma in the liver region fifteen years previously. At the operation for excision of the tumour, this was found to arise from "a cirrhotic band two inches wide" in the left lobe of the liver, presumably in the site of the old injury.

EXTENSION AND DURATION OF THE DISEASE.

Extension.—Most observers seem to be agreed that intrahepatic metastasis is of common occurrence, this no doubt being a consequence of the rich vascular and lymphatic supply of that organ. Apparently there is very early invasion of capillaries, giving rise to intravascular thrombosis, and this in its turn, though not invariably, causes rapid dissemination of malignant nodules within the liver. (Incidentally, many observers advance this as an argument against the theory of multicentric origin.)

In some cases the process of thrombosis may reach the inferior vena cava and thus involve the heart. Ewing stated that distant metastases are not very frequent, but are most commonly found within the thorax.

This comparative freedom from metastasis, in spite of early thrombosis, may probably be a result of the extreme rapidity of tumour growth, and Karsner has commented on the usual absence of necrosis in the tumour thrombi, with consequent improbability of malignant embolism. On the other hand, he believed that distant metastasis occurs more frequently and with a wider distribution (due to vascular spread) than is commonly conceded.

Interestingly enough, Ewing quotes many authors who agree that these extrahepatic metastases may secrete bile.

Duration.—All workers (Counseller and McIndoe, Karsner, Hale-White,²⁰ Fried, et al.) refer to the shortness in duration of this disease. Karsner considered that the average duration is about six months, the exact time of onset being difficult to determine on account of pre-existing symptoms due to cirrhosis.

In Counseller and McIndoe's series of 5 cases the duration varied from 19 days to 3 years and 7 months. Hale-White stated that "after the appearance of a malignant tumour of the liver life is seldom prolonged over four months". Fried concluded from the literature that the disease "runs a rapid course and is rarely of more than three months' duration".

It has been maintained that the shortness of duration is due to the rich blood-supply of the organ. This not only provides ready access to the general circulation for the toxic products of the tumour, but also leads to early intrahepatic metastases with consequent interference with the metabolic functions of the liver—"the king of organs" (Boyd⁵).

Allbutt and Rolleston,¹ in their conclusion that the duration rarely exceeds six months, stated that this forms a striking contrast to secondary malignant disease of the liver, where the patient may often linger for a long while.

CAUSE OF DEATH AND ASSOCIATED DISEASES.

Cause of Death.—Death is usually due to a rapidly progressive malignant cachexia, but several observers have commented on the relative frequency of sudden death from severe intraperitoneal hæmorrhage, due to malignant erosion of a large vessel.

In some cases, however, as pointed out by Ewing, latent carcinoma has been found at the autopsy on patients who have died from other diseases.

Associated Diseases.—Tull⁴⁴ (1932), in an interesting study of 134 cases in Singapore, noted a frequent association with active pulmonary tuberculosis, and cholelithiasis, "but in none of these was the gall-bladder involved in the new growth".

SYMPTOMATOLOGY.

It is unfortunately true that primary carcinoma of the liver possesses neither a pathognomonic feature nor even a characteristic clinical picture. There may or may not be a history of vague gastro-intestinal symptoms, and the physical findings are always very variable, this probably depending to some extent at least on the mechanical effects of the growing tumour (Griffith¹⁹), and on its metastases in other organs. Clinically the usual picture

is simply that of portal obstruction of some kind, with its customary though varied manifestations.

According to Eggel's series of 164 cases, the chief symptoms appear in the following percentage incidence: Icterus, 61; ascites, 58.5; œdema, especially of the lower extremities, 41; splenic enlargement, 32; pyrexia, 14.

As regards the liver itself, Allbutt and Rolleston considered that this organ is usually enlarged and associated with local pain and tenderness, though rarely it may be reduced in size, owing to contraction of the coincident or antecedent cirrhotic tissue. Similarly in Tull's series, the liver was enlarged in 126 cases, but "definitely diminished" in 8. According to the former, the usual weight of the cancerous liver is about 125 oz., with extreme variations from 36 to 493 oz., though it is rare to find it weighing more than 200 oz. (The weight of the normal adult liver is 50 oz.) It was also their conclusion that the right lobe is more commonly involved than the left.

A very remarkable point is that though the typical anæmia and emaciation of malignancy usually develops *pari passu* with the new growth, rare cases have been referred to by Rolleston where the terminal exuberance of the neoplasm has been attended by a considerable gain in weight of the patient.

Enlargement of the spleen is an inconstant feature and variable in degree. If present it may be due rarely to metastasis, while Karsner believed that toxic factors together with general abdominal stasis contribute to its development.

It is rather surprising that definite splenic involvement is not more constant, when it is remembered that "even in health 25 per cent of the blood in the portal vein comes from the spleen" (Boyd), and some degree of portal obstruction must always be present, owing either to the neoplastic or cirrhotic elements, or to both. In any case splenic enlargement has usually only been noted post mortem, and it is doubtful if this feature could often be demonstrated clinically.

Recorded observations on the incidence of jaundice are interesting, in that, though often present, it is usually slight in degree⁴⁵ and late in onset. Thus Tull noted that "it is remarkable that one so often sees a liver actually riddled with new growth, and no jaundice". This forms a marked contrast with the prolonged dark staining so commonly found associated with secondary carcinoma of the liver, and it is possibly explained by the short clinical course of a primary neoplasm in this region.

The development of icterus may be attributed to mechanical factors, such as obstruction to the outflow of bile due to intrahepatic tumour nodules, or to the secondary involvement of the hilar lymph-glands, but also to the results of the cachexia and to the circulation of the toxic products of malignant growth.

DIAGNOSIS.

Most statistics on this subject are founded on observations at autopsy, and it appears obvious that the great majority of diagnoses are only made post mortem. In this connection Smith has pointed out that in tropical countries where primary carcinoma of the liver is a definite entity, it can be,

and is, readily diagnosed. Similarly, Strong and Pitts were able to make at least one correct ante-mortem diagnosis based on their previous experience with that class of patient. They stressed a point which they considered common and of diagnostic importance—namely, that the liver early becomes fixed, and “frequently enlarges upwards to a greater extent than downward”, and that this feature may easily be missed if the liver is merely palpated below the costal margin. In one of their reported cases the dome of the liver was demonstrated clinically and radiographically to extend up to the second rib.

Brulé⁷ (quoted by Counsellor¹⁰) also insisted on the significance of fixation of the liver “so that it moves neither with palpation nor with respiration”, and Tull observed that in 52 out of his 134 cases the dome of the liver was pushed up by the tumour “sometimes as high as the third rib”.

Von Glahn¹⁷ in 1924 remarked that on account of the undoubted association with cirrhosis any unusual clinical feature in such a case under observation should bring up the remote possibility of developing carcinoma. Thus if the cirrhosis were advanced to such a stage that a considerable decrease in the size of the liver would be expected, and if on the contrary the organ were enlarged or actually noted to increase under observation, there would thus be gained some slight evidence in favour of the rarer disease.

On establishment of a tentative diagnosis of carcinoma of the liver it is hardly necessary to stress once more the necessity of adequately excluding—as far as is possible—the presence of a remote primary neoplasm.

With regard to biochemical investigations, “the liver may be riddled with new growth and still function normally as judged by various function tests” (Tull, in 1932).

Again, clinically it would be impossible to differentiate a sarcoma from a carcinoma, but as regards pigmented growths the possibility of melanuria should be investigated.

OPERABILITY.

This aspect should always be considered, though unfortunately on account of the late development of symptoms, the rapid course of the disease, and its quick dissemination, operative measures can very rarely be practicable. Almost all the statistical details have been obtained from necropsy material and there are few records of operations for the removal of these tumours.

Keen²⁷ in 1889 had collected reports of 76 cases of operations on the liver for various conditions, including three primary carcinomata of his own, and concluded that “every case saving those manifestly beyond relief should be explored, and the later steps determined by what is found”.

With this belief Thompson⁴² in 1899 concurred, and noted with surprise the small operative mortality in the reports—6 deaths out of 38, and only 2 of these from hæmorrhage.

Anschütz² wrote a monograph on the subject from the operative standpoint in 1903, and both his and Thompson’s work was considered by Yeomans⁵³. He in 1915 was able to tabulate from the literature only 16 cases of operations for primary carcinoma of the liver. Out of these 16, 4 patients died from various causes within sixteen days, 6 patients had recurrences in periods varying from two months to eight years, while 6 patients were alive and well

from three to seven years subsequent to their operation. Griffith, in his review of 57 cases in childhood (1918), mentioned that laparotomy had been performed in only one of these, and that unsuccessfully; but considered that on account of the extreme gravity of the disease "it would seem justifiable to make such an effort at removal whenever in any way possible".

In 1923, at a meeting of the Royal Society of Medicine, 5 cases were discussed. Grey Turner presented particulars of an interesting case of excision of "an unusual type of hepatic adenoma" in a boy of 13, with recovery up to a period of twenty-two months after the operation. He gave a full description of his operative technique for partial hepatectomy, and mentioned that out of his long series of 14,923 operations he had only felt justified in performing the operation on two occasions. Garnett Wright^{50, 51} described a successful removal of a primary hepatoma in a man of 60. The operation, which was "of a somewhat incomplete nature", had not been followed by recurrence (within two and a half years), which fact he attributed to "low-grade malignancy, if any". Philip Turner reported an excision of a large hepatic adenoma which had undergone necrosis, and caused such a severe intraperitoneal hæmorrhage as to simulate a ruptured ectopic gestation and to precipitate rapidly a fatal post-operative issue. Frank Kidd also mentioned a case of "a rare form of hepatic adenoma" in a female of 57, which simulated subacute appendicitis with abscess formation. An apparently successful operation was unfortunately followed by the death of the woman from heatstroke. Nitch gave a brief summary of a case in which he had removed a "carcinoma of an adrenal rest" from a woman of 54, followed by death from recurrence in twenty months.

Although none of these 5 cases was a primary carcinoma, yet it is reasonable to conclude from them and from the other cases cited above that if an apparently primary neoplasm of the liver is encountered during the course of a laparotomy undertaken with some other diagnosis in mind, due consideration must be given to the possibility of its removal.

Although, on account of the essential nature of this disease, accurate and early diagnosis is rarely possible, it should be aimed at, and a process of exclusion applied in cases that do not present a definite and true clinical picture. For, as Yeomans stated in 1915, "the only hope for improvement in results lies in the direction of earlier operative exploration in patients with a tumour in the right upper quadrant, in whom no primary growth can be found elsewhere".

In the case of typical solitary adenoma at least—where "the benign character is due chiefly to encapsulation and transitional forms to adenocarcinoma occur" (Ewing)—an adequate excision of such should prove successful, and its results may provide a striking contrast to the alternative of surgical inactivity.

CASE REPORT.

R. B. F., married man, 51 years of age. The patient was referred for a further opinion by Dr. W. S. McDougall and by Mr. A. T. Moon, F.R.C.S., of Wallington, Surrey.

HISTORY (June 15, 1933).—The patient had noticed a lump in his abdomen seven days previously. During this week he had had vague discomfort all over

the abdomen, especially when lying on the right side. The discomfort had become an aching pain, both front and back, for two days. He said he had been examined for an insurance policy and passed as a first-class life on May 9, but apparently the abdominal examination was most cursory. There was no history of any previous illness for which he had been attended by a doctor since childhood complaints. He had never suffered from syphilis or lived abroad.

ON EXAMINATION.—The patient appeared a very fit man, not older than his years. Apart from the local abdominal condition, no other abnormalities were detected by any one of us. On abdominal examination a very large mass was both seen and felt occupying the

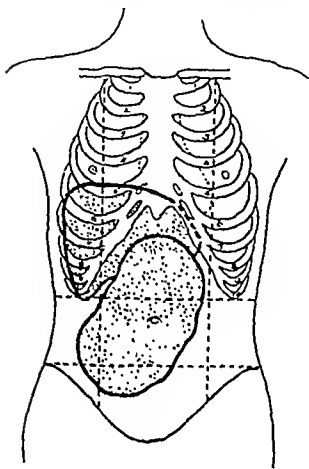


FIG. 421.—Diagram showing position occupied by tumour in abdomen.

centre and right side of the abdomen (Fig. 421). In the mid-line it extended from about an inch below the xiphisternum to about three inches above the symphysis pubis. Towards the left it extended to the mid-clavicular line, stopping short of the costal margin by about half an inch. To the right it extended well beyond the right mid-clavicular line into the right lumbar and right iliac fossa regions. Its shape appeared roughly that of a Rugby football, its main axis being directed from the left costal margin downwards to the right iliac fossa. Its surface appeared smooth, the edges shelving off with evidence of gross nodularity. In consistency it was very firm, with no sensation of fluctuation at all. The tumour moved very freely on respiration, and from side to side on manipulation. On percussion the tumour was quite dull, except at the right lower portion, where the colon slightly overlapped it. Above and to the left stomach resonance was easily detectable, causing a definite area of resonance between the tumour and the splenic dullness. The liver dullness extended from the upper border of the 5th rib in the right clavicular line downwards to just below the right costal margin. At this level the percussion note became slightly less dull for about one finger-breadth, and then again became

quite dull over the tumour. This small zone of impaired resonance between the normal liver and the tumour was presumably due to the fact that the duodenum, as shown by the X-ray examination, was lying behind the liver in this situation. Because of this physical sign, differential diagnosis from a pancreatic tumour was made difficult. There was no suspicion of ascites and no other abnormality could be detected in the abdomen. Rectal examination revealed no abnormality. The testes and prostate were normal. The urine contained no abnormality and the Wassermann reaction was negative.

An X-ray examination, including barium meal and barium enema skiagrams was kindly undertaken for me by Professor Morison. He reported as follows:—

Chest.—Revealed nothing abnormal; the levels of the diaphragm were normal, and its movements were free and unrestricted on both sides.

Barium Meal.—The body of the stomach was displaced to the left; the pyloric end and duodenum were displaced upwards. Nothing abnormal was detected in the stomach, the distortion being due to pressure from outside. The small intestine was displaced downwards.

Barium Enema.—The whole colon was outlined, the transverse colon being displaced downwards, the ascending portion of the transverse colon and splenic flexure were displaced to the left; nothing abnormal was detected in the colon, the displacement being due to the pressure from outside.

Findings.—The X-ray examination indicates the pressure of a large tumour which is displacing the body of the stomach to the left, the pyloric portion of the stomach and first part of the duodenum upwards, the descending portion of the duodenum to the right, and the transverse portion of the colon and small intestine downwards. The examination suggests a pancreatic tumour (which agreed with the clinical diagnosis).

Definite evidence of the hepatic origin of the tumour could probably have been determined by a hepatogram.*

OPERATION (June 20).—One hour before the operation the patient was given a hypodermic injection of one Roche's anipon containing omnopon gr. $\frac{7}{8}$, scopolamine gr. $\frac{1}{150}$. A spinal anæsthetic was skilfully administered by Dr. Ronald Jarman, 16 c.c. of 1-1500 perein being injected between the 3rd and 4th lumbar vertebrae. The patient was placed in the prone position for six minutes before being turned on to his back, all the time being in a slight Trendelenburg position. Nitrous-oxide-oxygen general anæsthesia was given by the McKesson apparatus. No chloroform or ether was used.

A long right paramedian incision revealed a large tumour arising entirely from the left lobe of the liver, which was displaced downwards and towards the right. The right lobe appeared quite normal, except for a small white spot about the size of a pin's head one inch to the right of the falciform ligament, and close to the antero-inferior edge. A careful search of the entire abdomen, including renal and suprarenal areas, failed to reveal any primary focus, and it appeared probable that the case was one of primary carcinoma of the liver, and it was decided to attempt removal.

Between the right lobe of the liver and the area in the left lobe occupied by the tumour was an interval of apparently normal left lobe of liver about one and a half inches in thickness.

Keeping close to the line of the falciform ligament, a series of catgut sutures, using double thickness No. 2 chromic catgut, were passed through the whole substance of the liver, and firmly tied. Each succeeding stitch slightly overlapped the preceding one, and working from below and above alternately, the tumour-bearing area, together with a good inch of apparently normal liver, was removed. This meant that practically the whole of the left lobe of the liver was removed. Little hæmorrhage occurred, several large arteries and veins being recognized and clipped before being cut across. To each of these a separate ligature was applied. A raw area some 7 by 3 in. remained which was still further drawn together by a thick running catgut suture, which incorporated the large hæmostatic sutures in its course. Complete hæmostasis was thus secured, and only a very small area about 3 in. by $\frac{1}{2}$ in. remained. The minute spot to the right of the falciform ligament was also removed with a small wedge of liver tissue. A cigarette drainage tube was inserted and the abdomen closed. Shock was inconspicuous. Apart from a slight temperature for a few days, the patient made an uninterrupted recovery, and has remained apparently perfectly well ever since.

PATHOLOGICAL REPORT.—The specimen (*Fig. 422*), when first removed, and still engorged with blood, weighed 5 lb. It was submitted to Dr. L. M. Hawksley, Pathologist at the Cancer Hospital, who reports as follows:—

An ovoid mass of tissue, $8\frac{1}{2} \times 6\frac{1}{2} \times 4$ in., weighing 3 lb. 11 oz.; liver substance obvious on the cut surface, the remainder showing a diffuse mottling of ill-defined nodules of new growth, which on section is seen to infiltrate the entire tissue, except for about 1 in. of apparently normal liver substance to the right of the tumour mass; the nodules range in size from that of a pin-point to one 2 in. in diameter, with cystic change.

Microscopic Examination.—Little variation in structure has been found in the numerous pieces taken for section. The thin fibrous tissue stroma is scanty, and it is a matter of difficulty to find even a few liver cells. The bulk of each section is made up of an epithelial tumour formed of tall and extremely regular columnar cells in tubular and glandular arrangement. Branching papillary processes project into cystic spaces, which contain also albuminous coagulum, and there is evidence of deposition of cholesterolin and of calcium salts. (*Figs. 423, 424.*)

Nodule from Right Lobe.—A small area shows a similar, but less advanced, condition.

* An intravenous injection of 20 to 25 c.c. of thorotrast daily for three days causes a deposit of thorium in the liver, spleen, and lungs, which then become opaque to X rays. Enlargements of liver and spleen or filling defects of these organs, or the presence of tumours in them or the lungs, are demonstrable. This method was not available in this case.

The histological features of the tumour indicate that it is an adenocarcinoma of the intrahepatic bile-duets, which has probably arisen in multiple foci representing a transition from simple adenomata. In view of the radical removal of the tumour-bearing tissue, the prognosis would seem to be influenced entirely by the tendency seen to the development of the same condition in the right lobe.

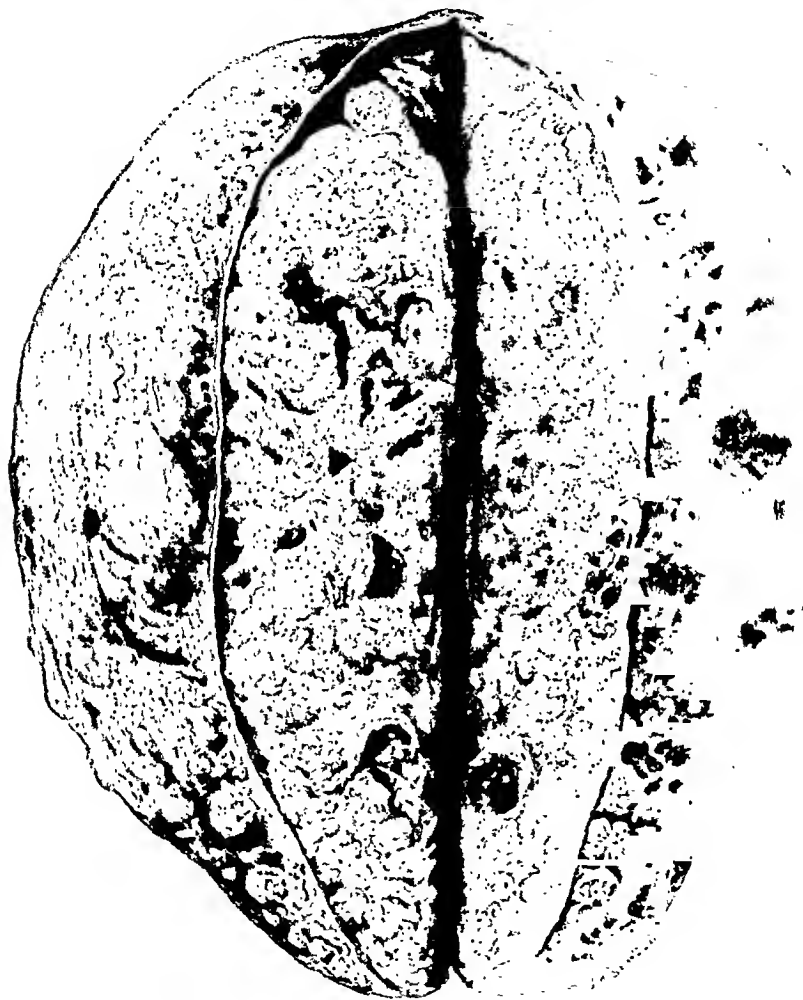
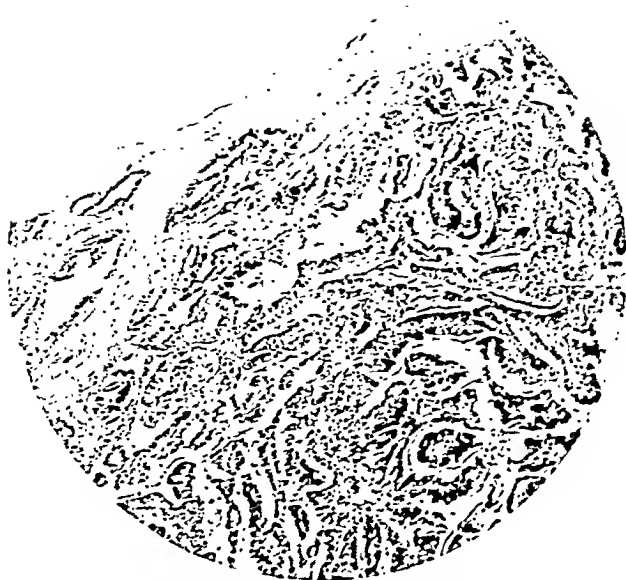


FIG. 422.—Tumour from in front, cut to display interior. ($\times \frac{3}{2}$.)

Professor Kettle was kind enough to examine the sections, and he confirms the pathological diagnosis in the following words:—

"I have been very interested to examine the sections of your liver tumour and agree with you as to its possible origin. The small nodule looks to me much more like a primary growth than a secondary deposit, and, although the main mass would do for an adenocarcinoma almost anywhere, I take it that it is probably a bile-duct carcinoma. I suppose there must be something disturbing the bile-duct epithelium through the whole liver, and it is quite likely that the big mass may represent several primary foci which have coalesced."



FIGS. 423.—Photomicrograph of a portion of the tumour. Immediately beneath the capsule of the liver the transition from simple dilatation of bile canaliculi to extensive proliferation of epithelium in single layers is seen. Throughout the greater part of the tissue the regular characters and adenomatous structure prevail. ($\times 40$.)



FIG. 424.—An area in which a papillary type of overgrowth has developed. In other parts the tumour tissue is of a more solid type, and small areas show calcium deposition and cyst-like spaces in which there is evidence of an accumulation of cholesterol. ($\times 40$.)

SUMMARY AND CONCLUSIONS.

1. Primary carcinoma of the liver is a rare disease, occurring chiefly in males, mainly between 40 and 60, though no age is exempt.

2. It has an average incidence of not more than 0·3 per cent of all autopsies.

3. There are two main types of tumour : (a) Hepatoma or carcinoma of liver cells ; (b) Cholangioma or carcinoma of intrahepatic bile-ducts.

4. Sarcoma of the liver occurs with even greater rarity, and clinically is indistinguishable from carcinoma.

5. The most commonly associated condition is cirrhosis, which is probably antecedent, and is indubitably related in etiology to carcinoma.

6. The relatively higher incidence in some other races is most likely attributable to various local parasitic infections of a chronic nature with consequent cirrhosis.

7. Diagnosis is difficult, though not impossible, and there are no pathognomonic features.

8. Fixation of the liver with upward enlargement (which may be demonstrated clinically, and confined radiologically) is sometimes present, while unusual features in a case of cirrhosis under observation may act as a signpost towards the correct diagnosis.

9. The prognosis is in all cases of the most extreme gravity, the disease running a rapid clinical course with an average duration of less than six months. In certain selected cases, however, it is not entirely without hope.

10. Intrahepatic metastasis is common. Extrahepatic metastases are less commonly encountered, and the mode of dissemination is almost certainly via the blood-vascular system.

In view of the liability to metastases, preliminary radiological examinations should be carried out.

11. The disease may be encountered during the course of a laparotomy (either exploratory or following a mistaken diagnosis). In each case a decision whether a radical partial hepatectomy can or cannot be undertaken must be made.

12. A case of massive cholangioma (carcinoma arising from intrahepatic bile-ducts) is fully described with details of a successful operation. Points of interest to be noted are :—

a. There was no general enlargement of the liver in an upward direction ; all the tumour enlargement had occurred downwards.

b. The tumour was freely movable both on respiration and from side to side on movement and on palpation.

c. Neither jaundice nor ascites was present.

d. The right lobe of the liver was almost unaffected, except for a minute nodule, microscopy of which led to an accurate diagnosis of the real nature of the main mass, which occupied almost the whole of the left lobe of the liver, and had grown to nearly twice the total weight of a normal liver.

e. The operation was not of great technical difficulty, and the patient made an uninterrupted recovery, and has remained well to the time of writing this article (January, 1934—seven months).

My thanks are due to Professor Kettle, Professor Morison, and Dr. L. M. Hawksley for permission to publish their reports, and also to Dr. W. Freeborn for his help with the extensive researches into the literature.

Note.—Since this article went to press the patient had a febrile attack with slight jaundice, from which he recovered, but on subsequent examination was found to have marked enlargement of his liver with ascites (March, 1934).

BIBLIOGRAPHY.

- ¹ ALLBUTT, C., and ROLLESTON, H., "Primary Carcinoma of the Liver". *System of Medicine*, 2nd. ed., 1908. iv, 215.
- ² ANSCHÜTZ, W., *Samml. klin. Fortr. Chir.*, 1903, No. 99.
- ³ ASCOLI, M., "Studio clinico sui Tumori primitivi del Fegato", *Policlinico (Sez. Chir.)*, 1930, xxxvii, 309. (Quoted by Brines.)
- ⁴ BLUMENAU, E., "The Cause of Death in Hepatic Cirrhosis", *Arch. f. Verdauungs-krank.*, 1920, xxvii, 1.
- ⁵ BOYD, W., *Surgical Pathology*, 1931, 421.
- ⁶ BRINES, O. A., *Amer Jour. Clin. Pathol.*, 1933, iii, 221.
- ⁷ BRULÉ, J., and CASTAIGNE, J., *Traité de path. Méd. de Thérap. appliquée*, 1923, xii, 383.
- ⁸ CASTLE, O. L., "Primary Carcinoma of the Liver in Childhood", *Surg., Gynecol. and Obst.*, 1914, xviii, 477.
- ⁹ CLAWSON, B. J., and CABOT, V. S., "Primary Carcinoma of the Liver", *Jour. Amer. Med. Assoc.*, 1923, lxxx, 909.
- ¹⁰ COUNSELLER, V. S., and MCINDOE, A. H., "Primary Carcinoma of the Liver of Possible Multicentric Origin occurring in a Case of Portal Cirrhosis", *Arch. of Internal Med.*, 1926, xxxvii, 363.
- ¹¹ DANCIE, C. B., "Primary Malignant Growth of the Liver in Infancy", *Lancet*, 1922, ii, 228.
- ¹² DOMINICI and MERLE, *Arch. de Méd. expér. et d'Anal. pathol.*, 1909, xxi, 136.
- ¹³ EGGEL, H., "Primary Carcinoma of the Liver", *Beitr. z. pathol. Anat.*, 1901, xxx, 506.
- ¹⁴ EWING, J., *Neoplastic Diseases*, 3rd ed., 1928. Philadelphia: W. B. Saunders Co.
- ¹⁵ FAIRLEY, Quoted by Fried.
- ¹⁶ FRIED, B. M., "Primary Carcinoma of the Liver", *Amer. Jour. Med. Sci.*, 1924, cxviii, 241.
- ¹⁷ GLAHN, W. C. von, and LAMB, A. R., "Primary Carcinoma of the Liver", *Med. Clin. N. Amer.*, 1924, viii, 29.
- ¹⁸ GOLDZIEHER, M., and BOKAY, Z. von, "Primary Carcinoma of the Liver", *Virchow's Arch.*, 1911, cciii, 75.
- ¹⁹ GRIFFITH, J. P. C., "Primary Carcinoma of the Liver in Infancy and Childhood", *Amer. Jour. Med. Sci.*, 1918, clv, 79.
- ²⁰ HALE-WHITE, Allbutt and Rolleston's *System of Medicine*.
- ²¹ HANOT, V., and GILBERT, A., *Etudes sur les Maladies du Foie*, Paris: Asselin & Houzeau.
- ²² HELVESTINE, *Jour. of Cancer Res.*, 1918, iii, 275.
- ²³ HEUKELOM, S. von, "Adenocarcinoma of the Liver with Cirrhosis", *Beitr. z. pathol. Anat.*, 1894, xvi, 341.
- ²⁴ HICKS, E. S., *Canad. Med. Assoc. Jour.*, 1929, Feb., xx, 169.
- ²⁵ JAFFE, R. H., "Sarcoma and Carcinoma of the Liver following Cirrhosis", *Arch. of Internal Med.*, 1924, xxxiii, 330.
- ²⁶ KARSNER, H. T., "A Clinicopathological Study of Primary Carcinoma of the Liver", *Ibid.*, 1911, viii, 238.
- ²⁷ KEEN, *Ann. of Surg.*, 1899, xxx, 267.
- ²⁸ KELSCH and KIENER, "Contribution à l'Etude de l'Adénome du Foie", *Arch. de Physiol.*, 1876, ii-iii, 622.
- ²⁹ LUBARSCU, *Zur Lehre von den Geschwulsten*, 1899.
- ³⁰ MANN, F. C., "Studies in the Physiology of the Liver", *Amer. Jour. Med. Sci.*, 1921, Jan., clxi, 37.
- ³¹ MUIR, R., "On Proliferation of the Cells of the Liver", *Jour. Pathol. and Bacteriol.*, 1908, xii, 287.

- ³² MÜLLER, W., "Primary Carcinoma with Senile Dementia and Old Echinococcosis. Case in Patient 83 Years Old", *Centralb. f. allg. n. pathol. Anat.*, 1932, Dec., lvi, 161.
- ³³ ORTH, *Pathologische Anatomie*, 1887.
- ³⁴ PIRIE, J. H., "Carcinoma of Liver in Natives", *Med. Jour. S. Africa*, 1921, xvii, 87.
- ³⁵ RIBBERT, H., "Malignant Adenoma of the Liver", *Deut. med. Woch.*, 1909, xxxv, 1607.
- ³⁶ ROWEN, H. S., and MALLORY, F. B., "A Multinucleated Liver-cell Carcinoma", *Amer. Jour. Pathol.*, 1925, i, 677.
- ³⁷ SABOURIN, C., "Contribution to the Study of the Lesions of the Liver Parenchyma with Cirrhosis", *Rev. de Méd.*, 1884, iv, 321.
- ³⁸ SALTYSKOW, "Carcinoma of Liver", *Verhandl. d. deut. path. Gesellsch.*, 1912, xv, 292.
- ³⁹ SCHMIEDEN, Quoted by Travis.
- ⁴⁰ SMITH, L. W., "Four Unusual Malignant Tumours of the Liver", *Arch. of Pathol.*, 1926, i, 365.
- ⁴¹ STRONG, G. F., and PITTS, H. H., "Primary Carcinoma of the Liver", *Arch. of Internal Med.*, 1930, xlii, 105.
- ⁴² THOMPSON, J. E., *Ann. of Surg.*, 1899, xxx, 284.
- ⁴³ TRAVIS, C. H., "A Case of Multiple Primary Adenocarcinoma of the Liver with Cirrhosis", *Bull. Johns Hopkins Hosp.*, 1902, xiii, 108.
- ⁴⁴ TULL, J. C., *Jour. Pathol. and Bacteriol.*, 1932, xxxv, 557.
- ⁴⁵ WARELEY, C. P. G., *Trans. Med. Soc. Lond.*, 1927, i, 124.
- ⁴⁶ WEGELIN, K., "Adenocarcinoma and Adenoma of the Liver", *Virchow's Arch.*, 1905, clxxix, 95.
- ⁴⁷ WHEELER, F. H., "Some Observations on Primary Carcinoma of the Liver", *Guy's Hosp. Rep.*, 1909, xlviii, 225.
- ⁴⁸ WINTERNITZ, M. C., "Primary Carcinoma of the Liver", *Johns Hopkins Hosp. Rep.*, 1916, xvii, 143.
- ⁴⁹ WOLLSTEIN, M., and MISELL, H. R., "Report of a Case of Hepatoma in an Infant", *Arch. of Pediatrics*, 1919, xxxvi, 268.
- ⁵⁰ WRIGHT, G., *Proc. Roy. Soc. Med. (Surg. Sect.)*, 1922-3, xvi, 56.
- ⁵¹ WRIGHT, G., *Lancet*, 1923, i, 333.
- ⁵² YAMAGIWA, K., "Contribution to the Knowledge of Primary Parenchymatous Carcinoma of the Liver (Hepatoma)", *Virchow's Arch.*, 1911, cxi, 437.
- ⁵³ YEOMANS, *Jour. Amer. Med. Assoc.*, 1909, lii, 1741; 1915, lxiv, 1301.

SHORT NOTES OF RARE OR OBSCURE CASES

SPHEROIDAL-CELL CARCINOMA OF BREAST: EXTENSION OF GROWTH WITHIN THE PERIMYSIUM.

By R. J. V. PULVERTAFT.

DIRECTOR OF PATHOLOGY, THE JOHN RUFORD CARLILL PATHOLOGICAL LABORATORIES,
WESTMINSTER HOSPITAL.

CONSIDERABLE practical, as well as theoretical, importance attaches to the method of extension of carcinoma of the breast. The conceptions of Handley with regard to lymphatic permeation are well known, and to some extent regulate current operative practice in the removal of the pectorals, to ensure removal of the fascial planes in which lymphatics travel.

Histological evidence of the tendency of carcinoma to spread along channels, such as lymphatics, blood-vessels, and inside the sheaths of nerves, is quite definite. Direct involvement of voluntary muscle is, however, rare, and even when a carcinoma is clinically attached to the muscles, microscopic evidence of their invasion is very frequently lacking.

The very unusual picture shown in the accompanying illustrations (*Figs. 425, 426*) indicates direct permeation within the perimysium.

HISTORY.—The history of the patient, a female, aged 54, married with one child of 20, was as follows:—

1917.—Suspected pulmonary tuberculosis.

1929.—Carcinoma of right breast: radical amputation.

1931.—Laryngitis; cause never definitely diagnosed.

1932.—Marked deterioration in general health, with difficulty in swallowing, sore throat, and cough.

1933.—Two secondary deposits in the scalp removed. During the last twelve months the patient lost $1\frac{1}{2}$ stones in weight. Persistent nausea and vomiting.

ON EXAMINATION.—When first seen on June 8 there was no evidence of any disease in the nose, throat, or neck. The vocal cords moved freely, the arytenoids were normal, and there were no enlarged cervical glands. There were two secondary deposits on the front of the chest, one over the sternum near the opposite breast, and the other at the level of the 9th rib in the mid-axillary line. These deposits were not attached to the skin, but clinically were fixed to the chest wall.

X-ray examinations showed no œsophageal obstruction and no metastases in the lungs, but some adhesions in the region of the diaphragm on both



FIG. 427.—Showing lower end of femur after removal. The tumour tissue can be seen after the condyles have been further separated through the line of fracture. ($\times 4$.)



FIG. 428.—Skiagram taken on Aug. 14, 1931, prior to operation. The destruction of bone by the tumour is shown and the line of fracture both transversely and into the joint is seen.

Fig. 428 shows the tumour and pathological fracture prior to operation (August, 1931), and *Figs. 429, 430* show the condition in June, 1933, when



FIG. 429.—Antero-posterior skiagram after reconstruction (June 28, 1933).



FIG. 430.—Lateral skiagram after reconstruction (June 28, 1933).

union is firm. It will be noted that the lower half of the patella is united firmly, but the upper half is standing away from the femur.

TWO CASES OF ILIOPSOAS BURSITIS.

By J. S. RAMAGE and G. BROWN MORTON, STOKE-ON-TRENT.

As this is a comparatively rare condition, and one which may cause considerable difficulty in diagnosis, the following two cases are perhaps worthy of record.

Normally there exists between the iliopsoas muscle and the ligamentum iliofemorale, a large bursa which frequently communicates with the synovial cavity of the hip-joint. The bursa lies in the line of the external iliac and

femoral arteries and is separated from them by the iliopsoas muscle. It will be readily understood that when it is enlarged and filled with fluid the overlying structures will be pushed forwards, and, on account of the relationship with the arteries, inguinal aneurysm will be closely simulated. As a matter of fact, in the cases here reported this resemblance was very close, and in the first case operation was carried out on a provisional diagnosis of aneurysm.

Case 1.—Male, aged 60 (1926). Shoemith.

HISTORY.—For the past eighteen months the patient had noticed a gradually increasing swelling in his right groin, which had recently begun to interfere with his work as a shoemith.

ON EXAMINATION.—There was a swelling the size of a large orange centred midway between the anterior superior iliac spine and the symphysis pubis, and lying chiefly above Poupart's ligament. The swelling was moderately tense, smooth, and rounded, and had ill-defined but regular margins. There was pulsation, apparently of expansile nature, synchronous with the arterial pulse. The pulse below the swelling was smaller than that of the opposite limb. No tenderness present. The range of movement of the right hip-joint was slightly diminished.

In view of these clinical findings, plus the fact that there was no evidence of syphilis or spinal caries, a provisional diagnosis of aneurysm of the external iliac artery was made.

OPERATION.—The external iliac artery was exposed by the extraperitoneal route, using an incision one inch above and parallel with Poupart's ligament. The external iliac vessels and the anterior crural nerve were identified. A large tense fluctuant swelling was defined between the iliacus and psoas muscles, pushing the nerve and blood-vessels forwards against Poupart's ligament. The nerve was gently retracted outwards, and the artery and vein inwards. The swelling was then aspirated by an exploring needle, and the diagnosis of iliopsoas bursitis made.

As much as possible of the wall of the bursa was excised, and the small posterior portion, which communicated with the hip-joint, was cauterized with carbolic acid. The wound was closed without drainage.

Convalescence was uneventful.

Case 2.—Male, aged 72 (1932). Gardener.

HISTORY.—Six months prior to admission to hospital this man noticed a swelling in his left groin. This caused no inconvenience whatsoever. Within the last few weeks, however, the swelling increased so rapidly in size that he called in his doctor.

ON EXAMINATION.—A swelling in the left groin was revealed. This was of elongated oval shape, about the size of a large lemon, and situated chiefly below Poupart's ligament, its axis being in the line of the external iliac and femoral arteries. The swelling was moderately tense, its outline regular, and its margins were rather ill-defined. Expansile pulsation synchronous with the arterial pulse was present. There was a slight impulse on coughing felt in the part of the swelling below Poupart's ligament. The range of movement of the left hip-joint was normal. There was no evidence of syphilis or spinal caries.

In view of our knowledge of the previous case, a diagnosis of iliopsoas bursitis was made.

OPERATION.—The external iliac artery was exposed as in the operation for *Case 1*, and the inner end of the incision was carried downwards along the axis of the swelling so as to expose the femoral sheath for a distance of two inches. A practically similar state of affairs was found as in *Case 1*. The swelling extended rather further below Poupart's ligament and had a bilobulated formation (*Figs. 431, 432*). The larger of the two sacs was actually above Poupart's ligament, although the external appearance had suggested the converse. The impulse on coughing was no doubt due to a fluid wave from the larger to the smaller sac. No communication with the hip-joint could be demonstrated.

The same surgical treatment was adopted as in *Case 1*.

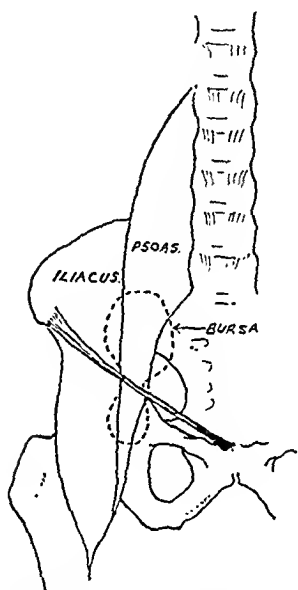


FIG. 431.—*Case 2*. Diagram to show approximate extent of bursa.

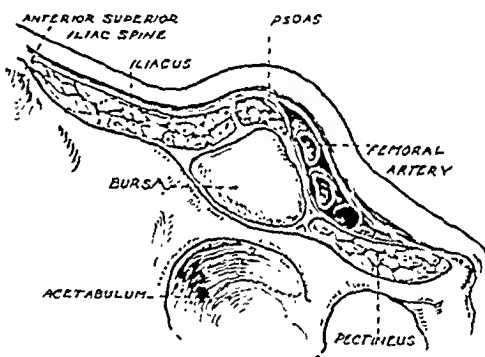


FIG. 432.—*Case 2*. Diagram to show relations of bursa.

COMMENTS.

In each case a definite cause for the enlargement of the bursa could be found—namely, in *Case 1* the patient was a shoemaker, and was in the habit of holding the horse's hoof in the right groin while hammering in the nails. In *Case 2* the patient had been doing a considerable amount of digging in a hard clay soil and was accustomed to strike the spade with his left foot. The swelling in the groin had developed only since his constant work in this hard soil. Factors of a chronic irritative nature were therefore present in each case, and were no doubt the cause.

The rarity of iliopsoas bursitis is no doubt due to the depth of its situation and corresponding freedom from trauma. When it does develop, its anatomical situation makes the diagnosis extremely difficult. It can therefore be easily understood that a considerable enlargement of the bursa with the transmitted pulsation of the overlying artery can closely simulate inguinal aneurysm.

cause of epilepsy is dealt with up to the incidence of epilepsy in brain tumours and its dependence on the site of the neoplastic lesion. There are 938 references at the end of the etiological section, and this is actually very valuable because the title of each paper is printed in full. The pathological part is well illustrated with microphotographs of Professor Ostertag's sections and Spielmeyer's work on the cornu ammonis, and the influence of vascular changes in bringing about focal degenerations is well described. More figures of glial alterations in large-scale pictures would have been welcome here.

The surgeon turns with especial interest to the last section of the book, "Physiologische Bemerkungen", for in this Krause describes his method of cortical stimulation. In a large-scale chart he records the motor points as observed in 142 patients whose Rolandic areas he has stimulated by the unipolar method. One reads with interest of Krause's conversations with Hitzig and of his reason for preferring the faradic to the galvanic current. Precise details are given of Krause's method of stimulation, current strength, and narcosis; it is surprising that he is not more favourable to local anaesthesia. Finally, there are adequate descriptions of stimulations of the general sensory and visual cortex, with remarks on the other special senses.

The reader closes the book with the feeling that here is gathered together all that is best of the world's thought on the various forms of epilepsy, and great credit is due to the guides who have led the reader so surely through such a maze. No English monograph exists which at all resembles it, for the English genius is more individual and more personal, and English readers may regret the care with which its authors have refrained from more passionate self-expression. Nor does the book contain a well-detailed account of local cortical excisions, and we should have welcomed Krause's mature opinion upon their results.

Vol. 48, which is of a quite different kind, must not detain us long. Its sections are written by several authors. All of these are excellent, with numerous original and striking illustrations. The sections deal with affections of the meninges (by Professor Guleke), cerebral softening and vascular degenerations (by Professor Boshamer), brain abscess (by Professor Willich), and a very well written article on otological and rhinological brain complications by Gustav Brühl, of Berlin. This last is what reviewers love to call a "veritable mine of information", and in this case the phrase is entirely applicable and true. Several sections by Hans Smidt, of Bremen, on vascular affections (sinus thrombosis, subdural haematoma, aneurysms) bring the volume to a close.

Human Embryology and Morphology. By Sir ARTHUR KEITH, M.D., F.R.S., LL.D., D.Sc., F.R.C.S., Master of the Buckton Browne Research Farm. Fifth edition. Medium 8vo. Pp. 558 + viii, with 535 illustrations. 1933. London: Edward Arnold & Co. 32s. 6d. net.

Books written by Sir Arthur Keith are always interesting, and his *Human Embryology and Morphology* has made a place among medical text-books which is unique. Keith approaches embryology from a different standpoint from that which is generally adopted. He begins the description of a system or organ by giving a brief outline of its evolution and comparative anatomy. This is followed by a short account of its development in the human subject. He has endeavoured to provide a concise account of human development which will be useful to students who are to become practical physicians and surgeons rather than to provide advanced students or anatomists with a complete account of recent research work. A treatise of the latter type could not at the present time be adequately dealt with in a single volume, nor could it be accomplished by a single individual. Keith has, however, introduced a new feature into the fifth edition: he has attempted to indicate the lines along which research is tending, by giving a selected list of references to recent work on each system and region, with short explanatory notes which follow his description in the text.

Some processes of development—more especially the histological changes which take place in the development of blood and lymph and in the growth of tissues such

as cartilage, bone, and nervous tissue—we should like to have seen treated in greater detail, both with regard to the text and in the technique of the illustrations. At the present time this is especially desirable in view of the increased interest which is being taken in the results of experimental embryology and tissue-culture—for example, the relation of the normal processes of cell production and growth to heredity, certain morbid conditions of the system and tissues, the growth of tumours, and the repair of injuries to bone. In many cases where recent work is at variance with old-established doctrines, the reader would be glad to have some comments or discussion on the points at issue—for example, the rôle played by the periosteum and osteoblasts in the formation of bone. This function has been challenged by both British and French authors. Is the surgeon to be guided in his methods of treatment by the more recent doctrine of the non-participation of the periosteum in the formation of bone, or is he to hold on to the older views? A brief discussion of the pros and cons of this and other questions would have been most valuable.

The book is very clearly written and the numerous illustrations are a valuable help to the comprehension of the descriptions in the text. Some excellent new illustrations have been added in this edition, and we draw attention especially to the type of drawing which on account of its being an attempt towards a faithful representation of reality is so much more convincing than a diagrammatic figure, which may be very misleading. We should like to mention especially *Fig. 359*, the endothelial lining of the heart in a 4th week human embryo (Veit and Eesch), and *Figs. 428 and 429*, representing models of the genital system of the female human fetus at the 7th and 10th weeks of development (R. H. Hunter). Modifications of original drawings, on the other hand, such as in *Figs. 18 and 27*, are not permissible, and may give rise to a wrong impression. Again, such a diagram as *Fig. 75*, which has obviously not been drawn from a dissection of the part, represents the internal intercostal nerve superficial to the internal intercostal muscle instead of deep to it, and affords an excellent example of the liability to error that occurs in any attempt to reconstruct nature on preconceived ideas or prevalent but incorrect notions. In reviewing the book as a whole, however, such faults as we have drawn attention to appear trivial, and one is most favourably impressed by the wonderful way in which an extensive subject has been tersely described and illustrated within the limits of a single volume, every page of which is interesting and informative.

Fractures. By PAUL B. MAGNUSON, M.D., Associate Professor of Surgery, Northwestern University Medical School, Chicago. Medium 8vo. Pp. 466 + xx, with 317 illustrations. 1933. Philadelphia and London: J. B. Lippincott Company. 25s. net.

THIS volume sets out to meet the needs of "the man who first sees a fracture", and after reading it one cannot come to any other conclusion than that the author has successfully arrived at his object. If the author's claim for it to be judged as a volume of personal experiences be admitted, criticism of it as a mirror of modern treatment of fractures is of little value; but if the general lines of treatment are compared with those in vogue in this country, it is apparent that the author places more reliance upon the use of wooden splints than has been customary here for the past fifteen or twenty years. The chapters on the principles of repair and of treatment are marked with sound common sense and are clearly the outcome of personal experience. A good deal of space is devoted to the general principles of operation on fractures. In our judgement this is superfluous if the book is for the use of general practitioners, and the pages devoted to operations scarcely suffice to provide a practical guide to anyone who has to undertake such operations. Again, where space is allotted to operative detail the selection of subjects dealt with scarcely follows any logical sequence, for we have a fairly complete account of the operative treatment of old fractures of the os calcis and innumerable operations for the correction of a fracture of the neck of the femur, yet no description is given of an operation for fracture of both bones of the forearm "because this is a formidable procedure".

The book is profusely and beautifully illustrated; in fact, it teaches more by its figures and pictures than by its letterpress. A certain number of the illustrations are scarcely worthy of production—for instance, the Sinclair skate, which has surely been superseded by the ice-tong caliper and the Steinmann needle, just as these have yielded place to Kirschner's wire; though in this respect the author has an opinion which may not be generally shared—his preference for the ice-tong caliper—and this for what we consider to be a completely inadequate reason—that it does not involve drilling through the bone.

Destructive criticism of this kind is not difficult in regard to any book which is a record of personal experience and opinion, yet, in spite of minor defects of this nature, this book can be recommended as a useful guide to any house surgeon setting out along the thorny path of fracture treatment, as well as to anyone who has had years of experience in this subject, for often the latter will find its perusal not devoid of suggestion and instruction.

Intracranial Tumours. By PERCIVAL BAILEY, Professor of Surgery, University of Chicago. Royal 8vo. Pp. 475 + xxiv, with 155 illustrations and coloured plates. 1933. London: Baillière, Tindall & Cox. 35s. net.

THOUGH this book is primarily a clinical study of the many pathological varieties of intracranial tumour, its outstanding feature is that Professor Bailey has arranged his material so as to set forth the evidence in support of the belief that a knowledge of the pathological nature of a cerebral tumour is of the utmost importance as a guide to its correct treatment. By approaching the subject from this aspect he has not only managed to throw fresh light upon many intricate problems of diagnosis, but has also drawn from his correlated clinical and pathological studies many deductions which are of value in prognosis. Though the localization of a cerebral tumour may still present considerable difficulty, modern aids to localization have reached such a degree of accuracy that the nature of the lesion becomes a matter of even greater importance than its position.

The first few chapters deal with the anatomical and physiological considerations which underlie the syndromes presented by cerebral lesions, and these are followed by a series of chapters every one of which is devoted to a single pathological entity. Accompanying the pathological picture is a description of the signs and symptoms which indicate the site of origin of the tumour. While the acoustic and hypophyseal tumours naturally lend themselves to such treatment, it is most interesting to see how Professor Bailey has managed to associate the various forms of glioma with the localizing syndromes of their usual site of origin. For example, with the medulloblastoma comes the description of the syndrome of the cerebellar vermis, and with the astrocytomas the syndromes of the cerebellar hemisphere and of the frontal lobe. Pathology and symptomatology are so skilfully mingled that the final product is not only clear and coherent but also makes a fascinating study of a subject which is never an easy one, and which is too often treated in a dull and uninspiring fashion.

A chapter devoted to miscellaneous and metastatic tumours is followed by a most important discussion of general and differential diagnosis, and the last chapter gives a brief but comprehensive account of the principles which underlie the treatment of intracranial tumours. Technique is not described in detail, but many valuable hints are offered to guide the surgeon when he encounters the problems which still have to be tackled even after the tumour has been found.

The book is illustrated by pen-and-ink drawings which have little artistic merit, but what they lose in beauty they gain in clarity. Professor Bailey has already achieved distinction for his cytological and experimental researches, and he now proves that his clinical observations have kept pace with his laboratory studies. His book is a storehouse of valuable clinical observations, and the style is entertaining because the material was originally arranged for clinical demonstrations in the University of Chicago. It will be appreciated by students working for the higher degrees and diplomas in both medicine and surgery. All those who intend to make a special study of neurology will be well advised to master its contents, and even the most experienced may dip into it with much profit.

Surgery of the Stomach and Duodenum. By J. SMILTON HORSLEY, M.D., F.A.C.S., LL.D., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va. Royal 8vo. Pp. 260, with 136 illustrations. 1933. London: Henry Kimpton. 35s. net.

THIS monograph represents largely the personal experience of a well-known American surgeon and one widely recognized as entitled to speak with authority upon the subject with which it deals. The embryology, anatomy, and physiology of the stomach and duodenum are first described, and a successful attempt is made to correlate the known facts of physiology with the symptoms of gastric and duodenal affections and with the principles of their operative treatment. Stress is laid upon the motor functions, since their derangement is responsible for probably 80 per cent of all gastric symptoms. The essential motor government of the stomach and duodenum is mainly intrinsic and is "myogenic" in origin (Alvarez), although extrinsic nerve connections exert a regulating influence. Emptying of both stomach and small intestine is due to a "gradient of contraction" (Alvarez), which consists in a more rapid movement of the muscular apparatus of the proximal than of the distal portion.

In the section upon etiology the importance of the contact of acid gastric juice with the intestinal mucosa, of streptococcal infection, and of the influence of the nervous system are all duly considered, and attention is directed to the alleged differences between America and Europe with regard to the type of gastroduodenal lesions. A study of 3000 case histories of gastroduodenal lesions operated upon at the Mayo Clinic shows that at the end of five years the death-rate in the cases of gastric ulcer was two and a half times that of the normal rate, while that in the cases of duodenal ulcer was but little above the normal.

In the preparation of patients for operation Horsley is a strong advocate of the continuous intravenous infusion of dextrose in Ringer's solution, and an excellent description is given of the technique.

The various operative procedures upon the stomach and duodenum are carefully and succinctly detailed, the sections dealing with gastrostomy, pyloroplasty, and partial gastrectomy meriting special mention. Horsley has a poor opinion of cholecystogastrostomy ("gastric bilification") in the treatment of gastric ulcer, while he favours the addition of resection of the gastric vagus nerves to such other local procedures as may be indicated in the treatment of cases of recurrent peptic ulcers, as tending to diminish reflex pylorospasm. Jejunal ulcer following gastroenterostomy is well described, as are the various procedures for its relief. The work is well illustrated, most of the illustrations being new, and there is a good index.

Die Bluttransfusion. By Prof. Dr. OEHLECKER (Hamburg). Super royal 8vo. Pp. 87 + viii, with 43 illustrations. 1933. Berlin and Vienna. Urban & Schwarzenberg. Paper covers, RM. 4; bound RM. 5.20.

THE author complains that previous books on this subject have dealt too much with the actual transfusion of blood, relegating to a secondary place the more important consideration, hæmolytic. He gives a short survey of the development of intravenous therapy, and then proceeds directly to this question of hæmolytic and allied reactions. Chapters follow describing the determination of groups, sources of error, and choice of donors, occupying about one-half of the book. There remain descriptions of various techniques and the indications for transfusion of blood.

Professor Oehlecker lays great emphasis upon the strict differences between hæmolytic and other reactions. Clinical evidence of hæmolytic always occurs one to two minutes after the commencement of the transfusion. He considers the signs and symptoms absolutely characteristic of this condition, and never to be confused with late reactions, frequently associated with rigors; these have no relation to hæmolytic, but to the donor's plasma or impurities in the citrate, etc. In consequence, prior to Landsteiner's discovery of the blood groups, Oehlecker devised his "biological test"—an assay of compatibility. This is now in constant use as an extra safeguard. The test is simplicity itself. Whilst an assistant retains his finger on the patient's pulse, the surgeon injects—intravenously—a small quantity

of the blood, 5 to 20 c.c., according to the condition of the patient. Some two minutes is allowed to elapse, and the procedure is repeated. In the absence of untoward symptoms the transfusion is completed in normal fashion. When incompatibility is present, the signs of hæmolysis will become apparent within the two minutes. They are akin to collapse, with pallor, falling-off of pulse, sweating, restlessness, and perhaps breathlessness. These somewhat alarming events rapidly subside without harmful effects. Before the introduction of blood grouping, which of course is now used as a routine measure in his clinic, Professor Oehlecker detected by this method among very many uneventful transfusions fifty cases of hæmolysis, with only one fatality. It appears therefore to be a reliable one, which may be adopted when a surgeon is driven to a transfusion without possessing means of grouping.

Some isolated points may be of interest. With an experience of 1000 transfusions, the author by no means objects to the repeated use of the same donor for the same recipient. Large quantities of blood are often of great value, from 1 to 1.5 litres. The professional donor in Munich receives 10 marks, in Berlin up to 20 marks, for each transfusion, and in New York 7 dollars per 100 c.c. of blood!

Of the various techniques, the indirect citrate method and his own are described in detail. The latter is a simple method by means of a syringe and two-way tap, of transferring whole blood directly from one person's vein to another, using intermittent irrigation with normal saline to avoid any clotting. It possesses the overwhelming disadvantage that on many surgical occasions close proximity of donor and patient is neither possible nor desirable. Professor Oehlecker draws his net far and wide in discussing the indications for transfusion. Few will differ from him, and it is interesting to note that in cases of acute staphylococcal or streptococcal septicæmia he does not consider a transfusion of any value; but of great help in the treatment of 'sepsis'. In acute pancreatic necrosis attention is drawn to the remarkable resuscitation which follows a large transfusion.

This short monograph is written in a clear style, in quite simple German, and is well worth reading. The author writes authoritatively from wide experience, and considers his subject from every aspect, supporting his arguments with personal cases and quotations from the literature.

Manuel de Radiodiagnostic clinique. By R. LEDOUX-LEBARD (Paris). Royal 8vo. In two volumes. Pp. 1075 + xvi, with 1143 illustrations. 1933. Paris: Masson et Cie. Fr. 260.

This is the best manual on radiology in the French language. It differs from other volumes in that art paper has been used throughout; consequently the radiographic illustrations, which have been well selected, are included in the text. It is not a mere atlas of pictures, but a descriptive account of the appearances which can be detected in disease on radiographic and radioscopy examination. The manual is published in two volumes and contains sections dealing with interpretation of radiographs and the localization of foreign bodies, the skeletal, nervous, circulatory, respiratory, digestive, and urinary systems, and the use of radiography in obstetrics and gynaecology. Unlike many former manuals on radiology, it does not contain long and tedious chapters on apparatus, which are for the most part culled from the pages of manufacturers' advertising publications. Some sections, notably those on the digestive, circulatory, and respiratory systems, are exhaustive and well illustrated. Interesting accounts and illustrations are given of the use of the more modern methods of radiographic investigation, including arteriography, ventriculography, pneumoperitoneum, cholecystography, intravenous pyelography, and lipiodol injections into the various tubular structures of the human anatomy.

The author and publishers are to be congratulated on the excellence of the illustrations and their reproduction. The volumes should be a valuable addition to the library of all radiologists.

Paralysis in Children. By R. G. GORDON, M.D., D.Sc., F.R.C.P. (Ed.), Physician, Bath and Wessex Orthopædic Hospital, etc.; and M. FORMSTER-BROWNS, M.D., M.S. (Lond.), Surgeon, Bath and Wessex Orthopædic Hospital, etc. Demy 8vo. Pp. 328 + viii. with 116 illustrations. 1933. London: Humphreys Milford. 15s. net.

THIS book represents the views of a neurologist and a surgeon, working together at an orthopædic centre for children where many cases of poliomyelitis and other paralytic conditions are admitted. The first part of the book deals with the general problems of paralysis, the physiology of movement, the location of the lesion which underlies the various syndromes described, the general pathology of paralysis, and the interpretation of symptoms. The second part is clinical and deals with muscular dystrophies, traumatic lesions of peripheral nerves, neuritis, juvenile tabes, anterior poliomyelitis, Landry's paralysis, Friedreich's ataxia, the cerebellar affections of childhood, disseminated sclerosis, striate lesions, spastic paralysis, chorea, and other types of paralysis. The third part deals with treatment, and is chiefly concerned with the various methods in the field of physiotherapy and non-operative orthopædies which have been used. Technical descriptions of operations are not given in any detail, except for some of the operations for the transplantation of tendons.

The book is certainly a most useful mine of information. Perhaps the sections on physiology are more profound than is necessary in a work of this kind. The chapters on re-education, the use of swimming exercises in warm water, and the adaptation of splints to paralysis are all most excellent, and the illustrations are very clear and well produced.

The Duodenum: Its Structure and Function, its Diseases and their Medical and Surgical Treatment. By EDWARD L. KELLOGG, M.D., F.A.C.S., Professor of Surgery and formerly Professor of Gastro-enterology, N.Y. Polyclinic Medical School, Imperial 8vo. Pp. 855 + xxvii, with 287 illustrations. 1932. New York: Paul B. Hoeber Inc. \$10.

THIS volume is described by the publishers as a monograph, but it is considerably bigger than a number of text-books devoted to the whole field of surgery. The author goes into very great detail, but nowhere does the subject seem to be unduly laboured. The work is based on twenty years of practice by the author and extracts from the literature, the extent of which can be judged by the fact that the references occupy 132 pages. The book commences with an account of the anatomy and physiology of the duodenum, together with chapters on its bacteriology and the methods used to examine it. The account of the X-ray examination is one of the best we have seen, but we cannot agree that a standard of perfection of such a degree has been reached that "it is unusual to demonstrate an ulcer at the operation table which the roentgenologist has failed to detect".

The first pathological condition to be dealt with is duodenitis, the importance and frequency of which is becoming more generally recognized. The author makes some interesting observations on the associated condition of duodenal glycosuria. Diverticula and duodenal ileus are well dealt with in separate chapters, and in both cases they are amplified by a number of clinical records.

Ulcer is naturally very fully considered, and it is a useful classification to look upon them as surgical, medical, and borderline cases. The methods of treating cases medically are considered in great detail together with the results which may be expected. It is a strong point in the book's favour that the author everywhere puts both sides of the case fairly and allows the reader to form his own judgements from them. We are glad to see that he deals with loss of liver dullness in perforation of ulcers by saying that it is probably rather infrequent. He goes on to qualify this by saying that it did not occur in a series of 100 cases on which he operated.

The surgical procedures are collected together into one chapter at the end of the book—an admirable arrangement which allows them to be referred to with the greatest of ease. They consist very largely of excellently reproduced drawings illustrating the operations, so that it is possible to follow them without referring to the text at all.

The production of the book is one on which the publishers are to be congratulated. The division of the index into subjects and proper names is perhaps a small point, but is one which makes for clearness. It is a most comprehensive book, and one which should be in the hands of every gastro-enterologist, whether physician or surgeon.

A Practical Medical Dictionary. By THOMAS LATHROP STEDMAN, A.M., M.D. Twelfth edition. Royal 8vo. Pp. 1256 + xii. Illustrated. 1933. London: Baillière, Tindall & Cox. 35s. net.

MEDICINE progresses rapidly, and doctors coin new words, which are often uncouth, more quickly than lexicographers can assimilate and explain them. No finality, therefore, is possible for a medical dictionary. Dr. T. L. Stedman, of New York, published the first edition of his *Practical Medical Dictionary* in 1908; the twelfth revised edition appeared in 1933. He has been fortunate in bringing so great a work to a successful issue. Mr. Henry Power and Dr. Leonard Sedgwick undertook a similar task for the New Sydenham Society in 1879. They carried it on courageously to the letter O, when the funds of the Society were exhausted and the rest of the alphabet had to be curtailed.

It is not possible to review a dictionary, but errors can be noted and suggestions offered for future editions. The errors are negligible. Thomas Bryant never received a knighthood, so the title appended to his name should be omitted. For additions it should be explained that 'brain' fever and 'low' fever were synonyms in the North of England for typhoid; 'Clutton's joints' should be mentioned, and it should be stated that 'Baker's cysts' are as often tuberculous as osteo-arthritis. 'Delbet's splint' as well as his sign should be described, and if 'Jackson's membrane' is mentioned, why is 'Lane's membrane' omitted? Aneurysms are 'wired' as well as bones, and to Woelfler's name should be added his 'islets'. 'Sciopody' is a convenient term for the monstrous growth of feet when children look as if they could shade themselves under their own soles. They were described by such old travellers as Sir John Mandeville long before they entered medicine. The recently published general index to the *BRITISH JOURNAL OF SURGERY* will provide Dr. Stedman with numerous additions to the thirteenth edition of his very useful and convenient dictionary, for the production of which we heartily wish him health and strength.

Surgical Anatomy. By GRANT MASSIE, M.B., M.S. (Lond.), F.R.C.S. (Eng.), Assistant Surgeon, Gny's Hospital, etc. Second edition. Medium 8vo. Pp. 458 + x, with 147 illustrations, many in colour. 1933. London: J. & A. Churchill. 18s. net.

THIS book certainly carries out what the author intended—"clothing the dry bones with life". The balance between anatomical detail and its surgical application is a happy one, and the subject matter seems well up to date.

We are not quite in agreement with the author on a number of subjects. The terminology used in describing the movements of the ankle is confusing. The description of the internal lateral ligament of the knee and the surface marking of the lateral sinus are both misleading. Many of the figures are not strictly accurate, such as that of the knee-joint (*Fig. 127*), in which the synovial membrane is represented as covering the articular cartilage. Other figures show so few points that it would seem unnecessary to include them. *Fig. 4*, for instance, representing sub-temporal decompression, whilst perhaps justifiable in a text-book of operative surgery, seems waste of space in a book of this sort. *Fig. 121*, too, shows very little relevant to the lumbar plexus that could not have been set out in a list giving the roots from which the nerves arise. The best section in the book is that on the abdomen, and it is here, too, that the best illustrations are to be found. The inclusion of a chapter on the autonomic nervous system is a unique and stimulating addition which should prove useful.

In spite of a few small inaccuracies in text and figures the author is to be congratulated on the production of a book which should be of great use to senior students and to those who, after qualifying, wish to refresh quickly their knowledge of anatomy.

The Operative Story of Cleft Palate. By GEORGE MORRIS DORRANCE, M.D., F.A.C.S. (Philadelphia), assisted by ENAYAT SHIRAZY, D.D.S. Medium 8vo. Pp. 561 + 8, with 534 illustrations. 1933. London and Philadelphia: W. B. Saunders Co. 32s. 6d. net.

No surgeon who is seriously interested in the surgery of cleft palate can afford to be without this excellent book by Dorrance, since nearly the whole of its 561 pages is devoted to a consideration of the literature of the subject. In a great many cases the original illustrations and some of the text of the various authors are copied *in extenso*. The magnitude of the labour can be judged from the bibliographical list, which runs into eighty-seven pages of the book, and it would appear as though the author had set himself the task of reviewing every paper that had ever been written on cleft palate.

The title of the book is somewhat misleading, in that the contents, although following a more or less chronological sequence, hardly constitute a story, but rather a complete catalogue—one which, nevertheless, is bound to remain for many years a standard work of reference.

It is only human that the author should finish his work with an account of his own operation, which, somewhat inappropriately, he styles the 'push-back' operation, whereas it is assumed that his aim is to free the soft tissues sufficiently to allow them to be pulled back by their own muscles; it is difficult to imagine soft tissues being pushed back. The push-back operation is strongly recommended by its author as the method of choice, but he has, unfortunately, followed a great many of those who write on this subject, and failed to record the number of cases on which he has operated, the mortality if any, the number of those in whom he has restored the palatopharyngeal sphincter and the evidence on which this is based, and, above all, the speech results and the standard of speech he regards as being good, bad, or indifferent; the example of Victor Veau must have been constantly before him since he uses some Veau methods.

The illustrations throughout are excellent, but unfortunately some of them cannot be accepted seriously, e.g., *Fig. 518* cannot by any stretch of imagination be said to present evidence of the restoration of the palatopharyngeal sphincter, since it was shown years ago that the appearance of the palate, as viewed from below, was no indication of velopharyngeal competence.

As a work of reference, the book is indispensable; but that part devoted to Dorrance's own opinions and methods requires more than his bald dogmatism before it can be generally accepted.

The Radiology of Bones and Joints. By JAMES F. BRAILSFORD, M.D.(B'ham), M.R.C.S. (Eng.), Radiologist, Queen's Hospital, Birmingham, etc. 9½ × 7 in. Pp. 500 + xx, with 310 illustrations. 1934. London: J. & A. Churchill. 30s. net.

This is a very valuable book, and in producing it Dr. Brailsford has made a most necessary contribution to radiology. Notwithstanding the title of the book it deals almost entirely with diseases of the bones and joints, fractures having been given very scanty consideration. This is probably a wise decision, because there are nearly five hundred pages. The author has discussed the various anatomical regions separately, and this will be found a great advantage in making references. Most conditions, both rare and common, have been dealt with as fully as possible, and a very full bibliography is given to enable a reader to make further study.

It is not a criticism to say that one would like to see a wider discourse on the radiographic manifestations of tuberculous affections of the joints, for this is a most important part of a radiologist's work and one which yields unsatisfactory results in a large number of cases. The recent work of Allison and Ghormley has emphasized the limitations of radiography in the diagnosis of joint disease.

The reproductions are plentiful, and on the whole good, but there are some which would have been more descriptive had they been larger. Dr. Brailsford is to be congratulated, and his work should find a wide circle of readers.

The Surgery of the Sympathetic Nervous System. By GEORGE E. GASK, C.M.G., D.S.O., F.R.C.S., Professor of Surgery, University of London, etc.; and J. PATERSON ROSS, M.S.(Lond.), F.R.C.S., Reader in Surgery, University of London, etc. Imperial 8vo. Pp. 163 + xii, with 13 plates and 30 other illustrations. 1934. London: Baillière, Tindall & Cox. 16s. net.

OUR only quarrel with this book lies in its title. An account of the many directions in which the surgery of the autonomic nervous system is going forward is hardly to be undertaken at this time and compressed within the scanty limit of 159 pages, however well written they might be. Subject to this limitation, and considering the fact that the authors' personal experience is confined to relatively few cases, the work has been extraordinarily well done. Regarded as a critical summary of the present state of our knowledge in the commoner applications of sympathectomy the book is admirable. The difficult subject of anatomy and physiology is clearly and thoroughly well described, whilst, following this, the preliminary investigations, so important before operation, are comprehensively and helpfully outlined. Throughout the remaining chapters, which deal with disorders of the circulation, disorders of the visceral motor mechanism, and with pain, the authors have given a summary in which the criticism is always keen and fair and the judgement exceptionally sound and restrained.

We note a tendency to regard the results of sympathectomy in thrombo-angiitis obliterans a little more hopefully than was done in the Bradshaw Lecture of 1932, and we would criticize the account of the parasympathetic supply to the distal colon as an inaccurate report of Stopford's original description.

In the surgery of the autonomic system the time factor is the great difficulty in the assessment of results. The present revival of interest in this branch must wait some years before definite statements can be made. The question of relapse is a very real one and further information is necessary on the recovery of intrinsic function of organs deprived of their sympathetic supply. These criticisms are not intended in any way to detract from the value of this book, and we should like to state how much we have appreciated the clear-cut and fair presentation which it gives. Any surgeon who may be contemplating an incursion into this difficult and largely uncharted field must have this book. He will find throughout its pages a guidance always safe and helpful.

Chirurgie infantile d'Urgence. By M. FÉVRE, Ancien Chef de Clinique chirurgicale infantile, Chirurgien des Hôpitaux de Paris. Preface by Professor OMBRÉDANNE. Large royal 8vo. Pp. 452, with 110 illustrations. 1933. Paris: Masson et Cie. Fr. 70.

THIS is a comprehensive volume dealing with every aspect of emergency surgery in childhood. In the main the teaching is similar to that practised in this country; there are, however, several exceptions. High tracheotomy is advised in all cases of obstruction, and an ancient type of coin-catcher is used to remove small coins from the œsophagus. This form of treatment has surely been replaced by direct removal by means of an œsophagoscope even in children. The author is very optimistic with regard to the treatment of the various kinds of spina bifida, the final result of which is too often hydrocephalus.

The illustrations are good on the whole, but it is a pity that a book devoted to emergency surgery in children should depict figures of adult patients. The index is small and incomplete for such a volume.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

- On Osteogenic Sarcoma. By JACON VAN DER SPEK (Amsterdam). Royal 8vo. Pp. 219. Illustrated. 1933. Kemink en zoon N.V.—Over Den Dorn—Utrecht.
- The Spread of Tumours in the Human Body. By RUPERT A. WILLIS, M.D., B.S., D.Sc., Melbourne, Pathologist to the Alfred Hospital and to the Austin Hospital for Chronic Diseases, Melbourne. Demy 8vo. Pp. 540 + x, with 103 illustrations. 1934. London: J. & A. Churchill. 25s. net.
- Infections of the Hand. By ALLEN B. KANAVILL, M.D., Sc.D., Professor of Surgery, North-western Medical School, Chicago. Sixth edition. Medium 8vo. Pp. 552 + xvi, with 216 illustrations. 1934. London: Baillière, Tindall & Cox. 30s. net.
- St. Bartholomew's Hospital Reports. Edited by LORD HONNER, R. G. CASTLE, W. SNOW, C. F. HARRIS, H. H. WOOLARD, R. C. ELSLIE, W. G. BAL, G. EVANS, and J. P. ROSS. Vol. LXVI. Demy 8vo. Pp. 363 + xxiv. Illustrated. 1933. London: John Murray. 21s. net.
- L'Artériectomie dans les Artérites oblitérantes. By RENÉ LERICHE, Professeur à la Faculté de Médecine de Lyon; and PIERRE STRICKER, Ancien Chef de Clinique à la Faculté de Médecine de Strasbourg. Large 8vo. Pp. 198, with 78 illustrations. 1933. Paris: Masson et Cie. Fr. 40.
- Gelenksteifen und Gelenkplastik. By Professor Dr. ERWIN PAYR (Leipzig). Vol. I. 11 × 8 in. Pp. 880 + xiv, with 240 illustrations. 1934. Berlin: Julius Springer. Paper covers, RM. 120; bound, RM. 124.80.
- La Sténose hypertrophique du Pylore chez le Nourisson. By J. POUCH, Chirurgien des Hôpitaux de Marseille. 7 $\frac{3}{4}$ × 5 in. Pp. 108 + viii, with 16 illustrations in the text and 8 plates. 1934. Paris: Masson et Cie. Fr. 20.
- Lessons on the Surgical Diseases of Childhood. By WILLIAM RANKIN, M.B., Ch.B. Royal 8vo. Pp. 190. Illustrated. 1934. Glasgow: Alex. MacDougall. 21s. net.
- A Short History of Some Common Diseases. Edited by W. R. BELL, M.R.C.S., L.R.C.P. Demy 8vo. Pp. 211. London: Humphrey Milford. 10s. 6d. net.
- Il Cancro del Retto. By VITTORIO PETINARI. Crown 4to. Pp. 225 + iv, with 121 illustrations. 1934. Bologna: Lucio Cappelli. L. 30.
- Operating Room Procedure: For Nurses and Internes. By HENRY C. FALK, M.D., F.A.C.S., Clinical Professor of Gynaecology, New York University and Bellevue Hospital Medical College, etc. With a Foreward by EUGENE H. POOL, M.D., New York. Large 8vo. Pp. 413 + xxii, with 328 illustrations. 1934. New York and London: G. P. Putnam's Sons. \$3.00.
- Die moderne Finsenbehandlung. By DR. SVEND LOMHOLT (Copenhagen). Crown 8vo. Pp. 64. Illustrated. 1934. Berlin and Vienna: Urban & Schwarzenberg. 12s. 6d.
- Trattato di Patologia chirurgica generale e speciale. By Prof. OTTORINO UFFREDUZZI (Turin). Vol. II. Pp. 1199 + viii, with 25 plates and 448 illustrations. 1934. Turin: Unione Tipografico-Editrice Troninence. Lire 145.

INDEX TO VOLUME XXI

	PAGE		PAGE
ABDOMEN in hyperparathyroidism	21, 22	Ankle, Charcot's disease of - - -	481
ABEL, A. LAWRENCE: Primary carcinoma of liver: with report of a case successfully treated by partial hepatectomy - - -	684	— fracture of, avascularity and - -	471
Abcess of breast in ancient Egypt - -	387	— osteo-chondritis dissecans of - -	73
— cerebral, following lobectomy - -	287	Anomalous jejuno-ileal diverticula -	184
— of lung, lobectomy and - - -	278	Anteater, pharyngeal pouch in - - -	252
— psoas, calcification of - - -	464	Antimony tartrate in bilharzia bladder	633
— subphrenic, following lobectomy -	287	Antiseptics, urinary, prior to prostatec-	
— tuberculous, calcification of - 462,	464	— tomy - - - - -	441
Absence of diaphragmatic valves - - -	421	Appendicectomy, anto- - - - -	545
Achilles tendon, ossification of - 466,	491	Appendicitis, acute - - - - -	411
Achlorhydria after gastrectomy - - -	146	— — average stay in hospital after	
Acro-cyanosis - - - - -	115	— operation - - - - -	417
Adenoma, malignant, of prostate, with		— — expectant treatment, fallacy of	411
metastases simulating Pott's		— — — routine - - - - -	413
disease of spine - - - - -	677	— — in a femoral sac, with adventi-	
Adenomatous polyposis, cancer of gut		— tious vascularization of a se-	
and - - - - -	575	— gregated appendix - - - - -	544
Adenopapilloma of stomach with lymphoid hyperplasia of duodenum	519	— — immediate operation - - - -	414
Adhesions, pleural, experimental production in cats - - - - -	561	— — mortality of operation according	
— — in lobectomy - - - - -	264, 281	— to period since onset - - - -	415
Age, appendicitis mortality in relation to - - - - -	411, 417	— — — on different days of attack	411
— incidence of epidermoid cysts of		— — post-operative complications -	417
hands and fingers - - - - -	34	— — in relation to age and sex - 411,	417
— — jejuno-ileal diverticula - - -	187	Appendix, segregated, adventitious vascularization of - - - - -	544
— — primary carcinoma of liver - -	686	Arm, sympathetic innervation of - -	88
— — Raynaud's disease - - - - -	613	Arris and Gale Lecture: Pouches of the pharynx and œsophagus with special reference to the embryological aspects: R. W. Raven (Feb. 17, 1933) - - - - -	235
Albers-Schönberg's disease - - - -	481	Arteries, effect of sympathectomy on	
Amputation stumps, painful, sympathectomy in - - - - -	116	— 6, 9, 116, 604	
Anæsthesia in diaphragmatic hernia of infants - - - - -	430	— palmar, aneurysm of - - - - -	215
— lobectomy - - - - -	270, 279	— ulnar and radial, aneurysm of in palm - - - - -	217, 218
— local, for experimental paralysis of sympathetic system - - - - -	8	Arteriography in thrombo-angiitis obliterans - - - - -	607
— in pharyngoplasty - - - - -	349, 351	Arterioles and capillaries, effect of sympathectomy on - - - - -	11
— spinal, effect on barium enemata in megacolon - - - - -	16	Arteriovenous anastomoses, extra- and intradural - - - - -	230, 233
Anatomy of autonomic nervous system (see Nervous System)		Arthritis, cervico-thoracic sympathectomy in - - - - -	9
— peripheral sympathetic nervous system (see Sympathetic Nervous System)		Artificial pneumothorax in bronchiectasis - - - - -	260
Aneurysm of palmar arteries - - -	215	— — effect on pleural absorption rate	659
Angina pectoris, sympathectomy in -	18	— — prior to lobectomy - - - - -	279
Angiomata, calcification of - - - -	486	Astragalus, fracture of, avascularity and - - - - -	471
— intracranial and facial, associated -	229	— osteo-chondritis dissecans of - -	74
Animals, pharyngeal and œsophageal pouches in - - - - -	250	Atlas, dislocation of, spontaneous hyperæmie - - - - -	476
		Atony, muscular, in hyperparathyroidism - - - - -	21, 24

	PAGE		PAGE
Auricle, cervical - - -	176,	Blood-vessels, effect of sympathectomy	
Auto-appendicectomy - - -	545	on - - - - -	6, 9, 116, 604
Autonomic nervous system (<i>see</i> Nervous System; Sympathetic Nervous System)		— of extremities, innervation of - -	89
		— small intestine - - - - -	189
		Blue sclerotics in hyperparathyroidism - - - - -	22
		Bone changes in hyperparathyroidism	
		22, 462, 464,	500
		— fragments found in osteo-chondritis dissecans - - - - -	70
		— growth (<i>see</i> Calcification; Decalcification; Ossification)	
		— metastases, calcification and - -	480
		— — with papilloma of bladder - -	108
		— — to spine from malignant adenoma of prostate: Pott's disease simulated - - - - -	677
		— salt, chemical constitution of - -	463
		— shadows, radiological significance of density of - - - - -	467
		— tumours, decalcification in - -	468
		Bone-grafting in fractures - - -	474
		Boomerang needle-holder for prostatectomy with closure - - - - -	439
		Borborygmi in jejuno-ileal diverticula	200
		Brachialis anticus, avulsion of - -	490
		Bradshaw Lecture: 'The surgery of the sympathetic nervous system: G. E. Gask (December, 1932) - -	113
		Brain, abscess of, following lobectomy -	287
		— blood-vessel tumours of - - -	229
		— injuries in ancient Egypt - - -	3
		Branchial cartilage, persistent - 176,	182
		— cyst, distinction from fistula - -	176
		— fistulæ (<i>see</i> Fistulæ, Branchial)	
		— grooves and arches in embryos - -	173
		Breast, abscess of, in ancient Egypt -	387
		— cancer of right, followed by sarcoma of left after 18 years - - - - -	156
		— — spheroidal-celled: extension of growth within perimysium - - -	701
		— sarcoma of left, 18 years after carcinoma of right - - - - -	156
		Breasted's (J. H.) translation of Edwin Smith Papyrus - - - - -	2, 385
		Breathing rates, effect on pleural absorption - - - - -	652
		Brock, R. C.: Observations on pleural absorption - - - - -	650
		Bronchi, cancer of, lobectomy in - 278,	296
		Bronchiectasis - - - - -	257, 302
		— anatomy - - - - -	303
		— collapse therapy in - - - - -	257
		— congenital factors in etiology - -	302
		— drainage in - - - - -	260
		— etiology - - - - -	302
		— histopathology - - - - -	306
		— infection as a cause - - - - -	303
		— lobectomy in (<i>see</i> Lobectomy, Pulmonary)	
		— mechanical factors in etiology - -	302
		— pathology - - - - -	303
		— phrenic paralysis in - - - - -	257
		— physiology - - - - -	303
		— pneumothorax in - - - - -	260
BACILLARY dysentery, spontaneous dislocation of hip following	523		
<i>Bacillus welchii</i> in acute strangulation	597		
BAILEY, HAMILTON: The clinical aspects of branchial fistulæ - -	173		
Barium enemata in megacolon, effect of spinal anæsthesia on - - -	16		
BARRETT, N. R., and WHEATON, C. E. W.: The pathology, diagnosis, and treatment of congenital diaphragmatic hernia in infants -	420		
Bat, fruit, œsophageal pouch in - -	253		
Benzoate, sodium, prior to prostatectomy	441		
Bile-duct, common, cyst of - - -	536		
Bilharzia disease of bladder in England	632		
Bilharziasis, cancer of liver and -	689		
— hæmaturia due to - - - - -	54, 632		
Bladder, bilharzia disease of, in England	632		
— calculi of, hæmaturia with - -	52		
— — with Paget's disease - - -	480		
— cancer of, metastases and - 108,	112		
— closure of, with prostatectomy (<i>see</i> Prostatectomy)			
— diverticula of, hæmaturia with -	54		
— — prostatectomy in presence of -	450		
— effect of sympathectomy on - -	16		
— foreign body in, hæmaturia due to	55		
— over-distended, method of emptying	441		
— papilloma of, hæmaturia with	46, 48, 49, 50		
— — with widespread metastases, case report - - - - -	108		
— retractors used in prostatectomy with closure - - - - -	439		
— and ureter, innervation of - -	96		
— varicose veins of, hæmaturia from	55		
Blister test as guide to prognosis in cancer - - - - -	627		
Blood in bilharziasis - - - - -	635		
— calcium (<i>see also</i> Calcification)			
— — and phosphorus content in hyperparathyroidism - - - - -	501		
— chemistry in acute strangulation of intestine - - - - -	582		
— cytology as guide to prognosis in cancer - - - - -	627		
— dyscrasia, hæmaturia with - 56,	61		
— lipase test in prognosis of cancer -	627		
— transfusion after lobectomy - -	286		
— in urine (<i>see</i> Hæmaturia)			
Blood-flow, increase of, due to sympathectomy - - - - -	6, 9, 18		
Blood-supply, adventitious, to segregated appendix - - - - -	544		
— calcification of bone and - 466,	468		
Blood-vessel tumours of brain - - -	229		
Blood-vessels, diseases of (<i>see</i> Raynaud's Disease; Thrombo-angiitis Obliterans, etc.)			

	PAGE		PAGE
Bronchiectasis, study of the pathology of sixteen surgical lobectomies -	302	Calculi, renal, haematuria and -	51
— thoracoplasty in -	260	— — recumbency and decalcification -	480
Bronchopleural fistula following lobectomy -	287	— — and ureteric, of unusual size : repeated stone formation -	63
Bronchopneumonia following lobectomy -	287	— — ureteric, haematuria with -	52
Bronchoscopy and postural drainage, importance in lobectomy -	278	— — urethral, haematuria with -	52
BRYCE, A. GRAHAM : A contribution to the study of pulmonary lobectomy -	560	— — vesical, haematuria with -	52
Buerger's disease (<i>see</i> Thrombo-angiitis Obliterans)		— — with Paget's disease -	480
BURMAN, C. E. L. : Two cases of keloid formation, with comments -	527	Cancer of bladder, haematuria and -	46, 48, 49, 50
BURROWS, HAROLD : Some experimental observations bearing on the etiology of megacolon -	577	— — metastases and -	108, 112
— — The occurrence of serotal hernia in mice under treatment with œstrin -	507	— — breast, right, followed by sarcoma of left after 18 years -	156
BURROWS, H. JACKSON : Two cases of ossification in the internal semilunar cartilage -	398	— — spheroidal - celled : extension within perimysium -	701
Bursitis, iliopsoas, case reports -	705	— — bronchi, lobectomy in -	278, 296
BUTLER, H. B. : Repeated perforation of peptic ulcers -	145	— — colon, adenomatous polyposis and -	575
BUTLER, R. WEEDEN : Observations upon multiple intramesenteric diverticula of the small intestine -	329	— — infective nature of -	620
BUTLER, R. W., and MIMPRISS, T. W. : A case of hyperparathyroidism with certain unusual features -	500	— — keloid formation and -	527
BUXTON, ST. J. D. : Osteoclastoma of femur : excision : reconstruction operation -	703	— — of kidney (<i>see</i> Kidney, Carcinoma of) liver (<i>see</i> Liver, Carcinoma of)	
		— — multiple, of colon -	572
		— — — case reports -	573
		— — — conclusions -	576
		— — — discussion -	574
		— — — etiology -	575
		— — — etiology -	571
		— — — incidence -	570
		— — — prognostic guides -	627
		— — of prostate, haematuria and -	46, 49, 51
		— — rectum, adenomatous polyposis -	575
		— — rheumatism disappearing in -	627
		— — selenide treatment of (<i>see</i> Selenide Treatment of Cancer)	
		— — of stomach, recurrence twelve years after partial gastrectomy -	542
CÆCUM, enlargement of, following silica injections in rats -	577	Capillaries and arterioles, effect of sympathectomy on -	11
Calcification (<i>see also</i> Decalcification ; Ossification) -	461	Carcinoma (<i>see</i> Cancer)	
— of bone, Albers-Schönberg's disease and -	481	Cardiac pain, effect of sympathectomy on -	17
— — fragility of calcified bone -	480	Cardiospasm, œsophageal pouches with -	248
— — ischaemic, clinical significance -	480	Carpal scaphoid, fracture of, avascularity and -	469
— — Kienböck's disease and -	483	— — — ununited -	471, 482
— — Köhler's disease of metatarsal and -	482	— — semilunar, Kienböck's disease of -	483
— — metastases and -	480	Carpus, traumatic sclerosis and porosis of -	482
— — Paget's disease and -	481	Cartilage, branchial, persistent -	176, 182
— — Preiser's disease and -	482	— — fragments found in osteo-chondritis dissecans -	70
— — relation of vascularity to -	466, 468	— — semilunar (<i>see</i> Knee-joint)	
— — syphilitic osteitis and -	481	Cat, lumbosacral nerves in -	666
— — cavernous angioma -	486	— — mesenteric ganglion in -	673
— — hæmatomata -	484	— — pulmonary lobectomy in -	560
— — hydatid cyst of omentum -	532	Catheter drainage after prostatectomy -	451
— — intervertebral discs -	466, 488	— — indwelling, in prostatectomy with closure -	437, 441
— — pathological -	464	— — retained, preparation of, in prostatectomy -	449
— — clinical significance -	484	Causalgia, sympathectomy in -	12, 18, 116
— — pathology of -	461	Cerebral abscess following lobectomy -	287
— — physico-chemical mechanism -	463	— — thrombosis in ancient Egypt -	3
— — in Raynaud's disease -	486	— — tumours -	229
— — of semilunar cartilages -	466, 488	Cervical auricle -	176, 182
— — supraspinatus tendon -	466, 484	— — spine, dislocation of, in ancient Egypt -	386
Calcium and phosphorus balance in hyperparathyroidism -	26, 501	— — sympathetic system, anatomy of -	90

	PAGE		PAGE
Cervical vertebra, benign giant-cell tumour of - - -	513	Colon, cancer of, adenomatous polyposis and - - -	575
Cervico-thoracic ganglionectomy (<i>see also</i> Sympathectomy) 8, 605, 609, 614	614	— — multiple (<i>see</i> Cancer, Multiple)	
Charcot's disease of ankle - - -	481	— diverticula of, multiple, œsophageal pouches with - - -	247
Chemical constitution of bone salt - - -	463	— effect of sympathectomy on - - -	15
Chest wall, infection of, following lobectomy - - -	287	— hæmorrhage into, due to calcified cyst of omentum - - -	532
CHESTERMAN, JUDSON T.: Retrograde jejuno-gastric intussusception - - -	541	— hypertrophy of, hypogastric sympathectomy for - - -	129
Children, spontaneous dislocation of hip in - - -	523	— sympathetic innervation of distal part - - -	94
Cholangionia - - -	684	Comparative anatomy of sympathetic nervous system - - -	83
Chondroma of phalanx, bone-grafting in	475	Compound fractures, Winnett Orr treatment - - -	474
Chondrosarcoma of breast (left) 18 years after carcinoma of right - - -	156	Congenital coccygeal sinus (<i>see</i> Coccygeal Sinus)	
CHRISTOPHERSON, J. B., and WARD, R. OGIER: Bilharzia disease in England: the cystoscopic appearance of the bilharzia bladder before and after intravenous injections of sodium antimony tartrate - - -	632	— cystic kidney, hæmaturia with - - -	55
Chromaffin system, anatomy - - -	84	— diaphragmatic hernia (<i>see</i> Diaphragmatic Hernia)	
Cirrhosis of liver in etiology of hepatic cancer - - -	688	— factors in etiology of bronchiectasis	302
Clavicle, fracture of, in ancient Egypt	387	— pouches of œsophagus (<i>see</i> Oesophagus)	
Cleft palate - - -	347	— — pharynx - - -	236
— — causes of faulty speech in - - -	356	Constipation, hypogastric sympathectomy for - - -	129
— — defects in nasopharynx in - - -	348	Costal cartilages, calcification of - - -	466
— — mechanism of speech - - -	347	CRAN, BRENNAN S.: Enlargement of the anterior portion of the prostate gland - - -	453
— — pharyngoplasty in - - -	349	Craniotomy, osteoplastic, in intracranial hæmangioma - - -	230, 233
— — anatomical considerations - - -	349	Cricopharyngeus muscle, comparative anatomy of - - -	240
— — complications and dangers - - -	351	Crystalline silica injected into mesentery of rat's cæcum: megacolon and - - -	577
— — mortality - - -	353	Cystitis, hæmaturia with - - -	46, 48, 53
— — restoration of palatopharyngeal valve - - -	355	Cystoscopic appearances of bilharzial bladder - - -	632
— — results - - -	354	Cystoscopy in hæmaturia - - -	49, 53, 54, 55
— — as a secondary operation - - -	367	Cysts, branchial, distinction from fistulae - - -	176
— — technique - - -	349	— calcification of - - -	466
— — prognosis after operation - - -	362	— of common bile-duet - - -	536
— — repair of palate - - -	351	— gas, of intestine, case report - - -	539
— — 'snorting' test for competent palatopharyngeal valve - - -	355	— of hands and fingers, post-traumatic epidermoid - - -	29
— — speech training after operation - - -	364	— — — case histories - - -	29
— — types of cleft - - -	361	— — — clinical characteristics - - -	34
Coccygeal sinus - - -	219	— — — complications - - -	35
— — case reports - - -	225	— — — contents of cyst - - -	39
— — conclusions - - -	228	— — — historical account - - -	33
— — etiology - - -	223	— — — hypothesis of mode of formation - - -	39
— — histology - - -	221	— — — latent period between injury and development of cyst - - -	35
— — paraffin wax injection prior to operation - - -	226	— — — macroscopic appearance - - -	36
— — symptoms - - -	220	— — — microscopic examination - - -	36
— — treatment - - -	226	— — — origin of epithelium - - -	41
— — X-ray appearances - - -	220, 226	— — — summary - - -	42
Cœliac root of inferior mesenteric ganglion, anatomy - - -	669	— — — terminology - - -	29, 33, 34
COKKINS, A. J.: Multiple carcinoma of the colon: with four original cases - - -	570	— hydatid, of liver, calcification of - - -	465
Collapse therapy in bronchiectasis - - -	257		
Colles's fracture, disuse decalcification in	467		
Colloid R.A.S. in treatment of cancer (<i>see</i> Selenide Treatment of Cancer)			

	PAGE		PAGE
Cysts, hydatid, of liver, pedunculated -	147	Diverticula of jejunum-ileum (<i>see also</i>)	
— — omentum, calcification of -	532	Diverticula of Small Intestine -	183
— kidney, congenital, hæmaturia with -	55	— age incidence -	187
— mesenteric -	637, 647	— anatomy of small intestine and -	187
— as cause of intestinal obstruction -	104	— anomalous -	184
		— case report -	198
		— causation theories -	195
		— classification -	184
D ^{4S} treatment of cancer by -	619	— comparison with diverticula of	
D'ABREU, FRANK : A case of		large intestine -	183
lipoma of the mesentery -	212	— complications -	193
DEBENHAM, R. K. : An investigation of		— conclusions -	210
742 cases of hæmaturia -	44	— emptying of sac -	194
Decalcification of bone (<i>see also</i> Calcifi-		— enterogenous -	184
cation : Ossification) -	461	— experimental work -	209
— bone-grafting and -	474	— frequency -	186
— delayed union of fractures due to		— Meckel's -	184
- - - - -	467, 471, 482	— multiple false -	184
— disuse of limb causing -	467, 471	— pathology -	191
— hyperemic -	466, 468	— sex incidence -	187
— clinical significance -	471	— situation -	194
— Kümmell's disease and -	475	— stages in development of sac -	192
— non-union of fractures and -	471, 482	— symptoms -	199
— recumbency, and nephrolithiasis -	478	— treatment -	205
— spontaneous hyperæmic disloca-		non-operative -	207
tion of atlas and -	476	— X-ray aspect -	199, 203
— in tuberculous disease -	468	— Meckel's -	184
— tumours of bone -	468	— of œsophagus (<i>see</i> Oesophagus)	
— X-ray significance -	467	pharynx (<i>see</i> Pharynx)	
Deviation of septum in ancient Egypt -	385	— small intestine (<i>see also</i> Diverticula	
Diaphragm, absence of both valves -	421	of Jejunum-ileum)	
Diaphragmatic (<i>see also</i> Phrenic)		— multiple intramesenteric -	329
— hernia, congenital, in infants -	420	— acute complications -	336
— anaesthesia -	430	— case reports -	332, 336
— case reports -	424	— clinical state -	331
— development -	420	— diagnosis -	335
— diagnosis -	423	— etiology -	342
— laparotomy v. thoraco-		— experimental speci-	
tomy in -	430	mens -	343
— method of approach -	430	— general considerations -	329
— pathology -	421	— gross anatomy -	329
— pre-operative treatment -	429	— minute anatomy -	339
— reduction of -	431	— summary -	345
— summary -	433	— treatment of uncom-	
— treatment -	428	licated cases -	335
— types -	421	DODD, HAROLD : Pied forcé or march	
— X-ray aspect -	424, 428	foot -	131
DICKSON, W. E. CARNEGIE, and HILL,		Dogs, œsophageal pouch in -	253
T. R. : Malignant adenoma of		— used in experiments on acute strangulation of intestine -	584
the prostate with secondary		Drainage in appendicitis -	413
growths in the vertebral column		— bronchiectasis -	260
simulating Pott's disease -	677	— and bronchoscopy prior to lobectomy -	278
Digitalis prior to prostatectomy -	442	— catheter, after prostatectomy -	451
Dislocation of atlas, spontaneous hyper-		— in pulmonary lobectomy -	268, 284, 286
æmie -	476	DUFF, D. G. : Cyst of the common bile-	
Distoniasis, hepatic, cancer of liver		duct -	536
and -	689	Duodenal fossa of Treitz, inferior -	398
Diverticula of bladder, hæmaturia with	54	— hernia, right -	398
— prostatectomy in presence of -	450	— ulcer, perforation of, repeated -	145
— colon, multiple, œsophageal pouches		Duodenojejunal flexure diverticulum.	
with -	247	X-ray aspect -	204
— duodenum, first part -	155	Duodenum, diverticulum of first part -	155
— intestine, frequency -	186	— lymphoid hyperplasia of, with adeno-	
		papilloma of stomach -	511

	PAGE		PAGE
Duodenum obstructed by hydro-nephrosis - - -	151	Femur, plasmocytoma of, solitary -	373
Dysenteric synovitis, spontaneous dislocation of hip following - -	523	— slipped epiphysis of - - -	313
EARLY surgical cases: The Edwin Smith Papyrus - - -	1, 385	— — — case reports - - -	321
Editorial secretary, presentation to -	383	— — — endocrine dysfunction and -	318
Edwin Smith Papyrus - - -	1, 385	— — — etiology - - -	313
Egypt, surgery in, in 3000-2500 B.C. -	1, 385	— — — frame for treatment of -	320
Elbow-joint, osteo-chondritis dissecans of	72	— — — indirect trauma and - - -	318
Electrosurgery in intracranial hæmangioma - - -	233	— — — nursing of cases - - -	321
Embolie theory of causation of osteo-chondritis dissecans - - -	77	— — — summary - - -	327
Embryo, cricopharyngeus muscle in -	240	— — — survey of methods and results	326
Embryology of branchial fistulae - -	173	— — — X-rays showing progress -	322
— pharynx - - -	236	Fibroids, uterine, calcification of -	466
Embryonic origin of coccygeal sinus -	223	Fibroma of tibialis anticus, calcification of - - -	465
Empyema (<i>see also</i> Pleural Absorption) — following lobectomy - - -	287	Fibrositis ossificans progressiva - -	491
Endarteritis obliterans, sympathectomy in - - -	115	Fibula and tibia, fractures of - -	474
Endocrine dysfunction in etiology of slipped epiphysis of femur - -	318	Fingers, chondroma of, bone-grafting in - - -	475
Endotracheal anæsthesia in pharyngoplasty - - -	349, 351	— cysts of (<i>see</i> Cysts of Hands and Fingers)	
Enterogenous jejuno-ileal diverticula -	184	— in hyperparathyroidism - - -	22, 24
Epidermoid cysts of hands and fingers (<i>see</i> Cysts of Hands and Fingers)		Fistulae, branchial - - -	173
Epididymectomy in genital tuberculosis	456	— — — clinical features - - -	174
Epiphysis of femur, slipped (<i>see</i> Femur)		— — — complete extirpation of - -	179
'Epistaxis, renal' - - -	61	— — — conclusions on derivation from clinical study - - -	182
Epithelium, origin of - - -	41	— — — distinction from cyst - - -	176
Essential hæmaturia - - -	60	— — — embryology - - -	173
Eventration, or hernia through dome of diaphragm - - -	421	— — — the external opening - - -	174
Excretion urography in carcinoma of kidney - - -	394	— — — histology of excised tract - -	181
Eye, effect of sympathectomy on - -	14	— — — with internal orifice - - -	177
— sympathetic innervation of - -	86	— — — origin - - -	173
FACE in hyperparathyroidism - - -	22	— — — Rahl's theory of origin - - -	173
Facial and intracranial hæmangioma, associated - - -	229	— — — symptoms - - -	178
FAGGE, C. H. : A case of hydronephrosis	151	— — — Wenglowski's theory of origin	174, 182
FAIRBANK, H. A. T. : Osteo-chondritis dissecans - - -	67	— — — X-ray examination - - -	175, 177, 179
Falx cerebri, calcification of - - -	466	— — — bronchopleural, following lobectomy	287
FARMER, A. W., and LINELL, ERIC A. : A case of neurocytoma of the pelvic sympathetic trunk - - -	534	— — — coccygeal (<i>see</i> Coccygeal Sinus)	
Fat in pathology of intestinal diverticula - - -	191, 196	— — — œsophago-tracheal, pouch associated with - - -	243
Feet in hyperparathyroidism - - -	22	— — — vesico-vaginal, hæmaturia with -	56
Femoral hernia, strangulated - - -	601	FITZGERALD, H. WALDEN : Large calcified hydatid cyst of omentum impacted in the pelvis and causing obstruction of and hæmorrhage into the colon - - -	532
— sac, appendicitis in, with adventitious vascularization of a segregated appendix - - -	544	Flat-foot, pied forcé and - - -	140
Femur, fractures of neck, ununited - -	471	Foot, march (<i>see</i> March Foot)	
— — from plasmocytoma - - -	373	— — — mechanics of - - -	133
— — osteoclastoma of: excision: reconstruction operation - - -	703	Forearm, cavernous angioma of, with calcification - - -	486
		Foreign bodies in bladder, hæmaturia due to - - -	55
		Fossa intersigmoidea, hernia into, case report - - -	158
		Fouadin in billarziasis - - -	635
		Fractures of astragalus and malleolus, avascularity and - - -	471
		— bone-grafting in - - -	474
		— of carpal scaphoid, avascularity and - - -	469
		— — — ununited - - -	471, 482
		— cervical vertebra (third) - - -	513
		— clavicle in ancient Egypt - - -	387
		— Colles's, disuse decalcification in -	467
		— compound, Winnett Orr treatment -	474

	PAGE	SUBSTANCE, control of arteri-	PAGE
Fraetures, delayed union of, disuse de-		Holes and capillaries by	11, 12, 18
calcification causing -	471	Hæmangioma, intracranial and facial,	
— in etiology of osteo-chondritis dis-		associated -	229
sectans -	76, 78	Hæmatomata, calcification of -	484
— of femur neck, ununited -	471	— subperiosteal, ossification of -	489
— pathological : reconstruction		Hæmaturia -	44
operation -	703	— with bilharziasis -	54, 632
— from plasmocytoma -	373	— blood dyscrasia -	56, 61
— metatarsals in march foot 133, 136,	708	— cancer of bladder -	46, 48, 49, 50
— patella, wiring of, Lister and -	557	— kidney -	50, 391, 392
— renal calculi as late complication -	478	— prostate -	46, 49, 51
— of skull in ancient Egypt -	2	— causes -	49
— spontaneous, calcification and -	480	— with congenital cysts of kidney -	55
— of tibia and fibula -	474	— cystitis -	46, 48, 53
— ununited, immobilization and decal-		— cystoscopy in -	49, 53, 54, 55
cification in -	471, 482	— with diverticula of bladder -	54
Frame for treating slipped epiphysis		— essential -	60
of femur -	318	— with foreign body in bladder -	55
FRASER, IAN : The diverticula of the		— gravel -	52
jejuno-ileum -	183	— hæmophilia -	56, 61
Freiberg's disease of metatarsal -	483	— hydronephrosis -	56
FRESHMAN, EDGAR : A case of renal and		— importance as symptom -	59
ureteric calculi of unusual size.		— an investigation of 742 cases -	44
Repeated formation of calculi -	63	— with movable kidney -	56
Freyer's operation on prostate -	437	— nephritis -	52, 60
Fruit bat, œsophageal pouch in -	253	— as the only symptom -	46
		— with papilloma of bladder 46, 48, 49, 50	
		— polypi round internal urinary meatus -	54
		— prostatic enlargement -	46, 49, 51
		— prostatitis and prostatico-cystitis -	54
		— purpura of urinary tract -	55, 61
		— pyelitis -	46, 48, 52
		— pyelo-cystitis -	46, 48, 53
		— pyonephrosis -	54
		— review of literature -	59
		— with renal calculi -	51
		— summary of conclusions -	61
		— traumatic -	55
		— with tuberculosis of genito-urinary	
		tract -	48, 52
		— unexplained -	56
		— with ureteric calculi -	52
		— urethral calculi -	52
		— stricture -	54
		— urethritis -	54
		— varicose veins of bladder -	55
		— — — urethra -	56
		— various causes with severity of	
		symptoms -	45
		— in various decades of life -	46
		— with vesical calculi -	52
		— vesico-vaginal fistula -	56
		Hæmophilia, hæmaturia with -	56, 61
		Hæmorrhage following lobectomy -	272, 286
		— — prostatectomy -	452
		Hæmostasis in prostatectomy -	445
		HALLIWELL, A. C. : Right paraduodenal	
		hernia -	398
		Hands and fingers, cysts of, post-trau-	
		matic epidermoid (<i>see</i> Hands and	
		Fingers)	
		— in hyperparathyroidism -	22, 24
		HARRIS, S. HARRY : Prostatectomy with	
		closure : five years' experience -	434
G			
GANGLIA and rami, anatomy of -	90		
— inferior mesenteric, anatomy -	671		
— stellatum -	86, 91, 92		
Ganglionectomy, cervico-thoracic (<i>see</i>			
also Sympathectomy) 8, 605, 609, 614			
— lumbar (<i>see</i> also Sympathectomy)			
8, 604, 609, 614			
Gangrene of lung, lobectomy and -	564		
— sympathectomy in -	115		
Gas-cysts of intestine, case report -	539		
GASK, G. E. : A diverticulum of the			
first part of the duodenum -	155		
— — The surgery of the sympathetic			
nervous system -	113		
Gastrectomy in benign tumours of			
stomach -	521		
— gastric analysis after -	146		
— partial, recurrence of gastric cancer			
twelve years after -	542		
Gastric (<i>see</i> also Stomach)			
— analysis after gastrectomy -	146		
Gastro-hepatic ligament, tumour of -	638		
Gastrojejunal intussusception, retro-			
grade -	541		
Genital tuberculosis (<i>see</i> Tuberculosis)			
Geographical incidence of primary car-			
cinoma of liver -	685		
Glossopharyngeal nerve, placodal area			
of -	237		
Goitre, pharyngeal pouches and -	242		
Goose skin, sympathectomy and -	13		
Granuloma of intestine : stenosis of			
ileocecal valve -	370		
Gravel, hæmaturia with -	52		
Grawitz tumour of kidney -	388, 391		
Groves (E. W. Hey), presentation to -	383		
Guinea-pig, lumbar outflow in -	665		

	PAGE		PAGE
Haudek's niche, diagnosis from duodenojejunal flexure diverticulum	204	Hypogastric nerves, formation and distribution of - - -	96
Head in hyperparathyroidism - -	22	— — stimulation of, effect on bladder	17
— injuries in ancient Egypt - -	2	— sympathectomy for megacolon -	129
Heart pain, effect of sympathectomy on	17	Hypoglossal nerve, embryonic cervical vestigial structures and - -	239
Hepatectomy, partial, in primary carcinoma of liver - - -	695		
Hepatoma - - -	684, 687		
Hernia, diaphragmatic (<i>see</i> Diaphragmatic Hernia)		I	
— femoral, appendicitis in femoral sac	544	LEOCÆCAL valve, stenosis of -	370
— into fossa intersigmoidea, case report	158	Ileum, differences from jejunum -	190
— paraduodenal, right - - -	398	— diverticula of (<i>see</i> Diverticula)	
— serotal, in mice under treatment with oestrin - - -	507	— occluded by mesenteric cyst -	106
— strangulated - - -	601	Iliac fossa, left, retroperitoneal tumour of - - -	643
Herpetic vesicles, formation of, in causalgia - - -	12	Iliopsoas bursitis, case reports -	705
Hexamine prior to prostatectomy -	441	Immobilization of fractures, non-union and - - -	471, 482
HILL, T. R., and DICKSON, W. E. CARNEGIE: Malignant adenoma of the prostate with secondary growths in the vertebral column simulating Pott's disease -	677	— renal calculi due to - - -	478
Hip, dislocation of, spontaneous, in childhood - - -	523	In Memoriam:—	
— osteo-chondritis dissecans of -	72	Sir George Makins - - -	381
— tuberculosis of, decalcification in -	468	Indigo-carmin test prior to prostatectomy - - -	442
HOLT, ROBERT L.: The pathology of acute strangulation of the intestine - - -	582	Infants, diaphragmatic hernia in (<i>see</i> Diaphragmatic Hernia)	
Horner's syndrome - - -	87, 611	Innervation (<i>see</i> Nervous System; Sympathetic Nervous System)	
Horse, cesophageal pouch in - -	253	Instruments for prostatectomy with closure - - -	439
Humeri in hyperparathyroidism -	24	Intersigmoid fossa, hernia into, case report - - -	158
Hunterian Lecture: Wardill, W. E. M.: Cleft palate (Jan. 27, 1933) -	347	Intervertebral discs, calcification of 466, 488	
Hydatid cyst of liver, calcification of -	465	Intestinal obstruction from calcified cyst impacted in pelvis -	532
— — pedunculated - - -	147	— — mesenteric cyst causing -	104
— — omentum, calcification of - -	532	— — polyp, preaneurous nature of -	575
Hydronephrosis, hæmaturia with -	56	Intestine, diverticula of (<i>see</i> Diverticula)	
— unusual case - - -	151	— acute strangulation of - - -	582
Hyperæmia, decalcification and 466, 468, 471		— — — cause of circulatory collapse with strangulation of long loops - - -	585
Hyperæmic dislocation of atlas, spontaneous - - -	476	— — — death in - - -	582
Hyperealcæmia, metastatic calcification in - - -	464	— — — circulatory changes in strangulation of loops of moderate length - - -	587
Hypernephroma of kidney - - -	388, 391	— — — clinical considerations -	600
Hyperparathyroidism - - -	20	— — — commentary - - -	599
— bone changes in - 22, 462, 464, 500		— — — conclusions - - -	602
— calcium and phosphorus balance in - - -	26, 501	— — — evidence that toxic substances are present in fluid from a strangulated loop -	588
— case report - - -	20	— — — experimental - - -	583
— — — with unusual features -	500	— — — long loops of small gut -	584
— diagnosis - - -	26	— — — loops of moderate length -	587
— generalized osteitis fibrosa with muscular atony in - - -	21, 24	— — — nature of toxin produced in -	597
— nephrolithiasis and - - -	480	— — — origin of toxic substances present in exudate -	590
— operation for - - -	25	— — — pathology - - -	582
— renal function in - - -	500	— — — when total anæmia exists -	596
— symptoms - - -	20	— — — venous obstruction alone exists -	584
— X-ray appearances of bones in - 22, 26, 27, 501		— — — relation of toxæmia to cause of death in strangulation of long loops -	586
Hyperplasia, lymphoid, of duodenum with adenopapilloma of stomach	519	— — short loops - - -	595
Hypogastric nerves, anatomy - 98, 674		— gas cysts of - - -	539

	PAGE		PAGE
Intestine, granuloma of: stenosis of ileocecal valve - - -	370	Knee-joint in hyperparathyroidism -	24
— small, anatomy of - - -	187	— ossification in semilunar cartilage, external - - -	466, 492
— — diverticula of (see Diverticula)		— — internal - - -	404
Intracranial and facial hemangiomata, associated - - -	220	— — case reports - - -	404
Intramesenteric diverticula of small intestine (see Diverticula)		— — diagnosis - - -	409
Intra-urethral method of digital enucleation of prostate - -	437, 442	— — histological examination of specimens -	406
Intussusception, jejuno-gastric, retro-grade - - -	541	— — pathology - - -	409
'Ionization method' in cancer (see Selenide Treatment of Cancer)		— — summary - - -	410
Ipsissima Verba:—		— — symptoms - - -	408
— Lister and wiring the patella -	557	— — treatment - - -	409
Ischemic calcification of bone (see Calcification)		— osteo-chondritis dissecans of -	68, 75
		Köhler's disease of metatarsal -	483
		Kummell's disease of spine, decalcification and - - -	475
JANES, ROBERT M.: The surgical treatment of bronchiectasis -	257		
Jaw, lower, dislocation of, in ancient Egypt - - -	386	LANDZERT'S left paraduodenal fossa - - -	398
Jejunal ulcer, perforated - - -	145	Laparotomy v. thoracotomy in diaphragmatic hernia of infants -	430
Jejunogastric intussusception, retro-grade - - -	541	Lateral sinus thrombosis in ancient Egypt - - -	385
Jejuno-ileum, diverticula of (see Diverticula)		Lead selenide treatment of cancer -	619
Jejunum, differences from ileum - -	190	LAR, RALPH O., and BOWEN, KENNETH: An inquiry into the results of surgical treatment of genital tuberculosis in the male - -	456
— gas-cysts of, case report - - -	539	Leg, sympathetic innervation of -	102
JOHNSTONE, A. S.: Appendicitis in a femoral sac, with adventitious vascularization of a segregated appendix - - -	544	Leriche's sympathectomy (see Sympathectomy)	
Joints (see also under various joints)		Ligaments, calcification of - - -	466
— tuberculous, Robertson - Lavette operation for - - -	475	Ligation of vas prior to prostatectomy with closure - - -	441
JONES, R. WATSON, and ROBERTS, R. E.: Calcification, decalcification, and ossification - - -	461	Ligature carrier for boomerang needle-holder in prostatectomy - -	439
		LINELL, ERIC A., and FARMER, A. W.: A case of neurocytoma of the pelvic sympathetic trunk -	534
KELOID formation, case reports and comments - - -	527	Lipiodol injection into cervical fistula - - -	179
KEYNES, GEOFFREY, and TAYLOR, HERMON: A case of parathyroid tumour - - -	20	— — of coccygeal sinus - - -	220, 226
Kidney, carcinoma of - - -	388	Lipoma of mesentery, case report -	212
— — case report - - -	388	Lister and wiring the patella - -	557
— — diagnosis - - -	391	Liver, carcinoma of, primary - -	684
— — hæmaturia and - - -	50	— — — age incidence - - -	686
— — pathology - - -	391	— — — case report: cure by partial hepatectomy - - -	693
— — spinal metastases in - - -	389, 393	— — — cause of death and associated diseases - - -	690
— — treatment - - -	394	— — — classification - - -	687
— cysts of, congenital, hæmaturia with -	55	— — — diagnosis - - -	691
— movable, hæmaturia with - - -	56	— — — duration of disease - - -	690
— sarcoma of - - -	396	— — — etiology - - -	688
— tuberculosis of - - -	458	— — — extension of disease - -	689
Kienböck's disease - - -	483	— — — geographical variations -	685
KING, E. S. J.: Post-traumatic epidermoid cysts of hands and fingers -	29	— — — histogenesis - - -	687
Knee-joint, avulsion of quadriceps -	491	— — — historical survey - - -	684
— calcification of semilunar cartilages -	466, 488	— — — incidence - - -	685
		— — — operability - - -	692
		— — — pathology - - -	686
		— — — sex incidence - - -	686
		— — — summary and conclusions -	698
		— — — symptomatology - - -	690

	PAGE		PAGE
Liver, cirrhosis of, in etiology of hepatic cancer - - - - -	688	MACFARLANE, J. A., and LINNELL, ERIC A.: Benign giant-cell tumour of third cervical vertebra: a case report - - - - -	513
— cyst of, hydatid, calcification of - - - - -	465	MACLYN, D. J.: Neurosurgery in the treatment of diseases of peripheral blood-vessels - - - - -	604
— — — pedunculated - - - - -	147	Magill's (I. W.) endotracheal anaesthesia in pharyngoplasty - - - - -	349, 351
— melanoma of, primary - - - - -	688	Makins (Sir George), obituary notice - - - - -	381
— parasitic infections of, in etiology of cancer - - - - -	689	Malleolus, fracture of, avascularity and - - - - -	471
— sarco-carcinoma of - - - - -	688	Mandible, dislocation of, in ancient Egypt - - - - -	386
— sarcoma of - - - - -	687	Marble bones - - - - -	481
Lobectomy, pulmonary - - - - -	277	March foot - - - - -	131
— — adhesions in - - - - -	264, 281	— — — accounts in literature - - - - -	133
— — Alexander's technique - - - - -	560	— — — case report - - - - -	131
— — anaesthesia in - - - - -	270, 279	— — — — with signs of old and recent injury - - - - -	708
— — blood transfusion after - - - - -	286	— — — characteristics of 11 cases - - - - -	139
— — in bronchial carcinoma - - - - -	278, 296	— — — clinical features - - - - -	141
— — bronchiectasis - - - - -	261, 278	— — — descriptive summary - - - - -	141
— — — case reports - - - - -	287	— — — diagnosis - - - - -	142
— — — results - - - - -	273	— — — etiology - - - - -	141
— — — study of the pathology in sixteen cases - - - - -	302	— — — flat-foot in relation to - - - - -	140
— — — case reports - - - - -	287	— — — pathology - - - - -	142
— — — closure of chest - - - - -	264, 284	— — — treatment - - - - -	142
— — — pedicle - - - - -	282	— — — X-ray findings - - - - -	131, 141, 708
— — — complications - - - - -	272, 286	Meckel's diverticula - - - - -	184
— — — conclusions - - - - -	301	Megacolon, barium enema in, effect of spinal anaesthesia on - - - - -	16
— — — contribution to study of - - - - -	560	— — — experimental, observations bearing on etiology - - - - -	577
— — — drainage in - - - - -	268, 284	— — — hypogastric sympathectomy for - - - - -	129
— — — duration of operation - - - - -	285	Melanoma of liver, primary - - - - -	688
— — — essential principles - - - - -	278	Mesenteric cyst as cause of intestinal obstruction - - - - -	104
— — — experimental, discussion - - - - -	567	— — — ganglion, inferior, anatomy - - - - -	671
— — — effects of presence of necrotic lobe inside thorax - - - - -	564	— — — coeliac root of, anatomy - - - - -	669
— — — general description - - - - -	561	— — — tumours (<i>see</i> Paramesenteric Tumours)	
— — — production of pleural adhesions - - - - -	561	Mesenterico-parietal fossa of Waldeyer - - - - -	398
— — — summary - - - - -	569	Mesentery of caecum, silica injections into, in rats: and megacolon - - - - -	577
— — — third-stage operation, removal of necrotic lobe - - - - -	564	— — — lipoma of, case report - - - - -	212
— — — indications - - - - -	278	Metastases, bone, calcification and - - - - -	480
— — — incision for - - - - -	262, 280	— — — with papilloma of bladder, wide-spread - - - - -	108
— — — phrenic paralysis prior to - - - - -	279	— — — in retroperitoneal sarcoma - - - - -	645
— — — position of patient for - - - - -	280	— — — to spine in cancer of kidney - - - - -	389, 393
— — — — after operation - - - - -	271, 286	Metastatic calcification in hypercalcemia - - - - -	464
— — — post-operative treatment - - - - -	286	Metatarsals, Köhler's disease of - - - - -	483
— — — pre-operative considerations - - - - -	278	— — — in march foot - - - - -	133, 140, 708
— — — removal of lobe - - - - -	267, 281	Mice, scrotal hernia in, when under treatment with œstrin - - - - -	507
— — — shock and - - - - -	285	Micturition, effect of sympathectomy on - - - - -	16
— — — summary of cases - - - - -	301	MIDDLETON, D. STEWART: Occupational aneurysm of the palmar arteries - - - - -	215
— — — technique - - - - -	262, 279	MIMMIS, T. W., and BUTLER, R. W.: A case of hyperparathyroidism with certain unusual features - - - - -	500
— — — tourniquets used in - - - - -	264, 281		
Lumbar ganglionectomy (<i>see also</i> Sympathectomy) - - - - -	8, 604, 609, 614		
— outflow in guinea-pig - - - - -	665		
— retroperitoneal tumour - - - - -	643		
— spine, pain in, in cancer of kidney - - - - -	391, 392		
— splanchnic nerves, anatomy - - - - -	669		
— sympathectomy - - - - -	128, 609, 614		
Lumbosacral autonomic nerves in Macaque monkeys - - - - -	668		
Lung, abscess of, lobectomy and - - - - -	278		
— — — gangrene of, lobectomy and - - - - -	564		
Lymphadenitis, oesophageal pouches due to - - - - -	248		
Lymphoid hyperplasia of duodenum with adenopapilloma of stomach - - - - -	519		

	PAGE		PAGE
MOLLESWORTH, H. W. L.: Granuloma of intestine. Stenosis of ileocaecal valve	370	Nervous system, autonomic, anatomy of hypogastric nerves	98, 374
Monkeys, ericopharyngeus muscle in	240	inferior mesenteric ganglion	671
— lumbosacral autonomic nerves in	658	lesser splanchnic nerves	669
MONTETU, W. B. R.: A case of match foot (pied forcé) with signs of old and recent injury	708	pelvic nerves	675
Mortality after prostatectomy	435	plexus	100, 675
— in appendicitis (see Appendicitis)		radi	90, 667
— genital tuberculosis in male	458	sympathetic trunks	666
MORTON, G. BROWN, and RAMAGL, J. S.: Two cases of iliopsoas bursitis	705	nomenclature	665
Movable kidney, hæmaturia with	56	plan of the	665
MUR, E. G., and WENN-JOHNSON, A. E.: Adenopapilloma of the stomach associated with lymphoid hyperplasia of the duodenum	519	sympathetic (see Sympathetic Nervous System)	
Muscle, ericopharyngeus, comparative anatomy of	240	Neurectomy (see Sympathectomy)	
Muscles, avulsed, ossification of	490	Neurocytoma of pelvic sympathetic trunk	534
Muscular atony in hyperparathyroidism	21, 24	Neurosurgery (see Sympathectomy)	
— power, effect of sympathectomy on	14	NEWELL, R. L.: Coccygeal sinns	219
Myeloma of femur: excision; reconstruction operation	703	NICHOLS, M. F.: A case of vesical papilloma with widespread metastases	108
Myositis ossificans	490	Ninth nerve placodal area, ultimate fate of	237
		Non-specific foreign proteins in Iliergers' disease	607
		Nose, injuries to, in ancient Egypt	385
		NUTTALL, H. C. WARDLEWORTH: The fallacy of expectant treatment in acute appendicitis	411
NEEVUS, facial, associated with intracranial hæmangioma	229		
Nails in hyperparathyroidism	22	OBITUARY notice of Sir George Makins	381
Nasopharyngeal infections, dislocation of atlas due to	476	Occupational aneurysm of palmar arteries	215
— pouches in sloth bear and in pig	250	Oculomotor phenomena following sympathectomy	14
Nasopharynx defects in cleft palate	348	Œsophageal orifice in different species	240
Natives, keloid in	527	— — hernia in relation to	421
Neck, dislocation of, in ancient Egypt	386	Œsophago-tracheal fistula, pouch associated with	243
Necrotic pulmonary lobe, lobectomy and	564	Œsophagus, pouches of (see also Pharynx)	235
Needle and holder used in prostatectomy with closure	439	— — acquired, with cardiospasm	248
NELSON, H. P., and ROBERTS, J. E. H.: Pulmonary lobectomy: technique and report of ten cases	277	— — — due to adhesion with tuberculous lymph-glands	248
Neo-antimosan in bilharziasis	635	— — — of inflammatory origin	248
Neo-stibosan, failure in bilharziasis	635	— — — with obstruction at lower end	248
Neurectomy in carcinoma of kidney	394	— — — in animals	253
Nephritis, hæmaturia in	52, 60	— — — classification	235
Nephrolithiasis, recumbency, and decalcification	480	— — — congenital	243
Nerve, glossopharyngeal, placodal area of	237	— — — associated with defect in muscular coat	245
— hypoglossal, embryonic cervical vestigial structures and	239	— — — — diverticula of colon	247
Nerve-supply (see also Sympathetic Nervous System)		— — — — œsophago-tracheal fistula	243
— of œsophageal orifice	242	— — — due to imperfect separation of trachea and œsophagus	244
— small intestine	190	— — — in posterior wall	247
Nervous system, autonomic (see also Sympathetic Nervous System)		Œstrin, serotal hernia in mice under treatment with	507
— — — anatomy	664	O'MALLEY, M. G.: Acute infective osteomyelitis of the tibia: use of Winnett Orr technique	530
— — — — celiac root of inferior mesenteric ganglion	669	Omentum, calcified hydatid cyst of	532
		— great, tumour of	640

	PAGE		PAGE
Omnopon prior to antimony tartrate injections - - - - -	636	Papilloma of bladder with widespread metastases, case report - - -	108
Oreclideotomy in genital tuberculosis -	456	— renal papilla - - - - -	395
Orethido-vaso-vesiculectomy in genital tuberculosis - - - - -	457	— stomach, with lymphoid hyperplasia of duodenum - - - - -	519
Orr (Winnett) treatment of acute osteomyelitis of tibia - - - - -	530	Papyrus, Edwin Smith - - - - -	1, 385
— — — compound fractures - - - - -	474	Paraduodenal fossa of Landzert - - -	398
Os calcis, syphilitic osteitis of - - -	481	— hernia, right - - - - -	398
— — — tuberculosis of - - - - -	462	Paraffin method of cleansing diverticulum of bladder - - - - -	451
Ossification (<i>see also</i> Calcification; Decalcification)		— wax injection prior to operation on coccygeal sinus - - - - -	226
— of Achilles tendon - - - - -	466, 491	Paralysis from cord pressure in dislocation of atlas - - - - -	477
— myositis ossificans and - - - - -	490	— phrenic, prior to lobectomy - - -	279
— pathological, clinical significance -	489	— — in treatment of bronchiectasis -	257
— pathology of - - - - -	461	— of sympathetic system, experimental -	8
— in semilunar cartilage (<i>see</i> Knee-joint)		Paramesenteric cysts, case reports -	638
— subperiosteal - - - - -	489	— tumours - - - - -	637
Osteitis deformans, spontaneous fractures in - - - - -	481	— — classification - - - - -	647
— — vesical calculi with - - - - -	480	— — conclusions - - - - -	649
— fibrocystica, generalized, with hyperparathyroidism - - - - -	22	— — definition - - - - -	637
— syphilitic, bone changes in - - - -	481	— — diagnosis - - - - -	648
Osteoblasts and osteoclasts, functions of	461	— — discussion - - - - -	644
Osteo-chondritis dissecans - - - - -	67	— — historical account - - - - -	637
— of ankle - - - - -	73	— — incidence - - - - -	638
— — elbow - - - - -	72	— — prognosis - - - - -	649
— — embolic theory of etiology - - -	77	— — signs and symptoms - - - - -	648
— — etiology - - - - -	74	— — theories of origin - - - - -	647
— — of knee - - - - -	68, 75	— — treatment - - - - -	648
— — prognosis - - - - -	81	Parathyroid glands (<i>see</i> Hyperparathyroidism)	
— — summary - - - - -	81	Patella in etiology of osteo-chondritis dissecans - - - - -	75
— — traumatic origin of - - - - -	74, 76, 78	— fractures of, wiring of, Lister and -	557
— — treatment - - - - -	80	Pedunculated hydatid cyst of liver -	147
Osteoclastoma of femur: excision: reconstruction operation - - - - -	703	Pelvic nerves, anatomy of - - - - -	675
Osteogenesis <i>in vitro</i> - - - - -	475	— plexus, anatomy of - - - - -	100, 675
Osteomyelitis, acute, of tibia, Winnett Orr treatment - - - - -	530	— sympathetic trunk, neurocytoma of -	534
Osteoplastic craniotomy in intracranial hæmangioma - - - - -	230, 233	— viscera, effect of sympathectomy on sensation in - - - - -	17
Osteosclerosis fragilis generalizata -	481	Pelvis, calcified hydatid cyst of omentum impacted in - - - - -	532
		— in hyperparathyroidism - - - - -	22
		Perforation of peptic ulcers, repeated -	145
		Peri-arterial sympathectomy (<i>see also</i> Sympathectomy) - - - - -	115
PAGET'S disease, spontaneous fractures in - - - - -	481	Pericardium, calcification of - - -	466
— — vesical calculi with - - - - -	480	Perimysium, extension of cancer of breast within - - - - -	701
PAIN, A. B.: Recurrence of gastric carcinoma twelve years after partial gastrectomy - - - - -	542	Peripheral sensation, effect of sympathectomy on - - - - -	18
Pain in jejuno-ileal diverticula - - -	199	— vascular disease (<i>see</i> Raynaud's Disease; Thrombo-angiitis Obliterans, etc.)	
— lumbar spine in cancer of kidney -	391, 392	Peritoneal fluid in acute strangulation (<i>see</i> Intestine, Acute Strangulation of)	
Palate, cleft (<i>see</i> Cleft Palate)		— fosse, hernia into - - - - -	398
Palatopharyngeal valve, function in speech - - - - -	347	Peritonitis with appendicitis - - -	411
— — restoration of, in cleft palate -	355	Phalanges in hyperparathyroidism -	22, 24
Palmar arteries, aneurysm of - - -	215	Pharyngoplasty in cleft palate (<i>see</i> Cleft Palate)	
— aspect of hand, cysts in (<i>see</i> Cysts of Hands and Fingers)		Pharynx, development of - - - - -	236
Panner's disease of metatarsal - - -	483	— pouches of (<i>see also</i> Oesophagus) -	235
Papilloma of bladder, hæmaturia and	46, 48, 49, 50		

	PAGE		PAGE
Pharynx, pouches of, acquired, anterior	243	Preiser's disease	182
— — — posterior	239	Presacral nerves, anatomy of	98, 674
— — — in animals	252	formation and distribution of	96
— — — classification	245	stimulation of, effect on bladder	17
— — — congenital	236	Presentation to Editorial Secretary	383
— — — lateral	238	Prostate, cancer of, hæmaturia in	16, 49, 54
PHILLIPS, H. A. : Spindle-celled mesenteric tumours : with remarks on similar retroperitoneal tumours	637	— — — enlargement of anterior portion, case report	453
Phosphatase activity of tissues	464	— — — hæmaturia and	46, 49, 54
Phosphorus and calcium balance in hyperparathyroidism	26, 501	— — — in mice treated with oestrin	514
Phrenic (<i>see also</i> Diaphragmatic)		— — — malignant adenoma of, with metastases simulating Pott's disease of spine	677
— paralysis prior to lobectomy	279	— — — tuberculous of	157
— — in treatment of bronchiectasis	257	Prostatectomy, suprapubic, with closure	134
Pig, nasopharyngeal pouch in	250	— — — — — anterior transverse oblique sutures	448
Pied forcé (<i>see</i> March Foot)		— — — — — bladder retractors used in	439
Pilomotor mechanism, effect of sympathectomy on	13	— — — — — five years' experience	431
Pilonidal sinus (<i>see</i> Coccygeal Sinus)		— — — — — indigo-carminic test and	442
Placodal area, ninth nerve, ultimate fate of	237	— — — — — the indwelling catheter and need for asepsis	437, 441
Plasma phosphorus in hyperparathyroidism	26, 28	— — — — — instruments for	439
Plasmocytoma of femur, solitary	373	— — — — — intra-urethral method of digital enucleation of prostate	437, 442
Pleural absorption	650	— — — — — mortality	435
— — — absorption from inflamed pleura	655	— — — — — needle and holder used in	439
— — — — — uninfamed pleura	652	— — — — — posterior or trigonal suture	446
— — — effect of pneumothorax on rate of	659	— — — — — the postero-lateral hæmorrhagic sutures	445
— — — observations on	650	— — — — — post-operative catheter drainage	451
— — — — — practical applications	661	— — — — — hæmorrhage	452
— — — — — summary	662	— — — — — preparation of retained catheter	449
— — — adhesions, experimental production in cats	561	— — — — — preparatory treatment	440
— — — in lobectomy	264, 281	— — — — — in presence of diverticulum of bladder	450
Pleuro-peritoneal canal, hernia through (<i>see also</i> Diaphragmatic Hernia)	421	— — — — — reports of progress	435
Pneumothorax in diaphragmatic hernia of infants	430	— — — — — summary	452
— effect on pleural absorption rate	659	— — — — — surgical principles involved and operative requirements	438
— prior to lobectomy	279	— — — — — technique	442
— in treatment of bronchiectasis	260	— — — — — two-stage operation	451
POLSON, CYRIL, and SHIRES, N. : A solitary plasmocytoma of the femur	373	Prostato-cystitis and prostatitis, hæmaturia with	54
Polypi round internal urinary meatus, hæmaturia from	54	Protein therapy in Buerger's disease	607
Polyposis, adenomatous, cancer of gut and	575	Pseudo-coxalgia, X-ray appearances	73
Porosis and sclerosis of carpus, traumatic	482	Psoas abscess, calcification of	464
Post-traumatic cysts of hands and fingers (<i>see</i> Cysts of Hands and Fingers)		Ptosis following sympathectomy	14
Postural drainage and bronchoscopy prior to lobectomy	278	Pulmonary lobectomy (<i>see</i> Lobectomy, Pulmonary)	
Pott's disease of spine simulated by metastases from malignant adenoma of prostate	677	PULVERTAFT, R. J. V. : Spheroidal-cell carcinoma of breast : extension of growth within the perimysium	701
Pouches of œsophagus and pharynx (<i>see</i> Œsophagus ; Pharynx)		Purpura of urinary tract, hæmaturia with	55, 61
POWER, SIR D'ARCY : The Edwin Smith Papyrus	1, 385	PYBUS, F. C. : A case of gas-cysts of the intestine	539
— — — Ipsissima verba : Lister and wiring the patella	557	Pyelitis, hæmaturia with	46, 48, 52
		Pyelo-cystitis, hæmaturia with	46, 48, 53
		Pyelography in carcinoma of kidney	393
		Pyonephrosis, hæmaturia with	54

	PAGE		PAGE
Q UADRICEPS, avulsion of - - -	491	Raynaud's disease, calcification in -	486
Quadriplegia from cord pressure		— case reports - - -	614
in dislocation of atlas - - -	477	— — sympathectomy in 10, 120, 124,	614
R ABBITS, experiments on pleural		Rectum, cancer of, adenomatous poly-	
absorption in - - - - -	654	poidosis and - - - - -	575
Rabl's theory of origin of branchial		Recumbency, decalcification, and neph-	
fistulae - - - - -	173	rolithiasis - - - - -	478
Radial artery, aneurysm of, in palm -	218	REID, RONALD : Case of hernia into the	
Radium radiation and selenide treat-		fossa intersigmoidea - - - -	158
ment of cancer - - - - -	619	Renal calculi, hæmaturia and - - -	51
RAMAGE, J. S., and MORTON, G. BROWN :		— — recumbency and decalcification	478
Two cases of iliopsoas bursitis -	705	— — and ureteric, of unusual size :	
Rami, anatomy of - - - - -	667	repeated stone formation -	63
— and ganglia, anatomy of - - -	90	— ' epistaxis ' - - - - -	61
Ramsectomy, anatomical difficulties -	94	— function, failure of, in hyperparathy-	
Rare or Obscure Cases :—		roidism - - - - -	500
Appendicitis in a femoral sac, with		Repeated perforation of peptic ulcers -	145
adventitious vascularization of a		Respiration rates, effect on pleural ab-	
segregated appendix - - - - -	544	sorption - - - - -	652
Carcinoma of right breast followed		Retractors, bladder, used in prostatec-	
by sarcoma of left breast after an		tomy with closure - - - - -	439
interval of 18 years - - - - -	156	Retrograde jejuno gastric intussusception	541
Cyst of the common bile-duct -	536	Retroperitoneal hernia in intersigmoid	
Diverticulum of the first part of the		fossa, case report - - - - -	158
duodenum - - - - -	155	— lipoma, case report - - - - -	212
Gas-cyst of the intestine - - - -	539	— tumours (<i>see also</i> Paramesenteric	
Granuloma of intestine. Stenosis		Tumours) - - - - -	637
of ileocecal valve - - - - -	370	— — case reports - - - - -	640, 645
Hernia into fossa intersigmoidea -	158	Reviews and Notices of Books (Titles):—	
Hydronephrosis - - - - -	151	<i>Annals of roentgenology</i> , Vol. xiv	
Iliopsoas bursitis - - - - -	705	(L. Davis) - - - - -	554
Large calcified hydatid cyst of		<i>The art of surgery</i> (H. S. Souttar) -	549
omentum impacted into the pel-		<i>Atlante di Chirurgia dello Stomaco</i>	
vis and causing obstruction of		(G. Egidi) - - - - -	378
and hæmorrhage into the colon -	532	<i>Die Bluttransfusion</i> (Ochlecker) -	713
March foot (<i>pie'd forcé</i>) with signs		<i>Die Bruchoperation nach Bassini</i>	
of old and recent injury - - -	708	(A. Catterina) - - - - -	555
Neurocytoma of the pelvic sympa-		<i>Chirurgie infantile d'urgence</i> (M.	
thetic trunk - - - - -	534	Fevre) - - - - -	719
Osteoclastoma of femur : excision :		<i>Chirurgie du rectum</i> (H. Hartmann)	162
reconstruction operation - - -	703	<i>A clinical study of the abdominal</i>	
Recurrence of gastric carcinoma		<i>cavity and peritoncum</i> (E. M.	
12 years after partial gastrectomy	542	Livingston) - - - - -	169
Repeated perforation of peptic ulcers	145	<i>The cure of hæmorrhoids, varicose</i>	
Retrograde jejuno gastric intussus-		<i>veins and ulceration, and allied</i>	
ception - - - - -	541	<i>conditions</i> (S. McAnslaud) - -	377
A solitary plasmocytoma of the		<i>Demonstrations of physical signs in</i>	
femur - - - - -	373	<i>clinical surgery</i> (H. Bailey) -	547
Spheroidal-celled carcinoma of		<i>The diagnosis and treatment of pos-</i>	
breast : extension of growth		<i>trinal defects</i> (W. M. Phelps and	
within the perimysium - - -	701	R. J. H. Kipluth) - - - - -	378
R.A.S. treatment of cancer (<i>see</i> Selenide		<i>The digestive tract : a radiological</i>	
Treatment of Cancer)		<i>study</i> (A. E. Barelay) - - -	378
Rats, experiments on, in etiology of		<i>Discases of the spinal cord</i> (W. B.	
megacolon - - - - -	577	Cadwalader) - - - - -	378
RAVEN, R. W. : Pouches of the pharynx		<i>The duodenum</i> (E. L. Kellog) -	715
and œsophagus with special re-		<i>Die epileptischen Erkrankungen</i> (F.	
ference to the embryological and		Krause and H. Selum) - - -	709
morphological aspects - - -	235	<i>De l'excision haute de l'estomac</i>	
RAY, P. N. : Spontaneous dislocation of		(B.-O. Guiheneuc) - - - -	161
hip in childhood - - - - -	523	<i>Fractures</i> (P. B. Magnusson) -	711
Raynaud's disease - - - - -	613	<i>Handbuch der Unfallmedizin</i> (C.	
		Kaufmann) - - - - -	555
		<i>History of urology</i> (E. G. Ballenger	
		et al.) - - - - -	552

Reviews and Notices of Books, <i>contd.</i> —	PAGE	Reviews and Notices of Books, <i>contd.</i> —	PAGE
<i>Human embryology and morphology</i> (Sir A. Keith)	710	<i>The technic of local anaesthesia</i> (A. E. Hertzler)	553
<i>Idols and invalids</i> (J. Kemble)	167	<i>Treatato di patologia chirurgica generale e speciale</i> (O. Uffreduzzi)	165
<i>The injured workman</i> (G. F. Walker et al.)	377	<i>Treatment of fractures in general practice</i> (W. H. Ogilvie)	377
<i>Intracranial tumours</i> (P. Bailey)	712	<i>Die unspezifischen chronischen Erkrankungen der Wirbelsäule</i> (H. Burekhardt)	166
<i>Intracranial tumours roentgenologically considered</i>	554	<i>Urology in women</i> (E. C. Lewis)	167
<i>An introduction to 'avertin' rectal anaesthesia</i> (J. K. Mathlox)	163		
<i>Manuel de radiodiagnostic clinique</i> (R. Ledoux-Lebard)	714	Authors:—	
<i>The operative story of cleft palate</i> (G. M. Dorrance)	717	Bailey, H. : <i>Demonstrations of physical signs in clinical surgery</i>	547
<i>Operative surgery</i> (W. S. Bickham and C. M. Smyth, jun.)	518	Bailey, P. : <i>Intracranial tumours</i>	711
<i>Papers relating to the pituitary body, hypothalamus, and parasympathetic nervous system</i> (H. Cushing)	551	Bainbridge, W. S. : <i>Sixth international congress of military medicine and pharmacy, etc.</i>	169
<i>Paralysis in children</i> (R. G. Gordon and M. Forrester-Brown)	715	Ballenger, E. G., et al. : <i>History of urology</i>	552
<i>La phrénicectomie</i> (L. Bérard, F. Dumarest, and Desjaques)	168	Barelay, A. E. : <i>The digestive tract : a radiological study</i>	378
<i>The physician as a man of letters, science, and action</i> (T. K. Munro)	553	Bérard, L., et al. : <i>La phrénicectomie</i>	168
<i>Poliomyelitis</i>	376	Bickham, W. S., and Smyth, jun., C. M. : <i>Operative surgery</i>	518
<i>A practical medical dictionary</i> (T. L. Stedman)	716	Boschawer, K., et al. : <i>Die spezielle Chirurgie der Gehirnkrankheiten</i>	709
<i>The practice of surgery</i> (R. Howard and A. Perry)	518	Brailsford, J. F. : <i>The radiology of bones and joints</i>	717
<i>The principles and practice of rectal surgery</i> (W. B. Gabriel)	161	Brühl, G., et al. : <i>Die spezielle Chirurgie der Gehirnkrankheiten</i>	709
<i>The principles of treatment of muscles and joints by graduated muscular contractions</i> (M. Smart)	376	Burekhardt, H. : <i>Die unspezifischen chronischen Erkrankungen der Wirbelsäule</i>	166
<i>The radiology of bones and joints</i> (J. F. Brailsford)	717	Cadwalader, W. B. : <i>Diseases of the spinal cord</i>	378
<i>Recent progress in medicine and surgery</i> (ed. Sir J. Collie)	549	Callander, C. L. : <i>Surgical anatomy</i>	550
<i>Roentgenographic studies of the urinary system</i> (W. E. Lower)	379	Case, J. T. : <i>Annals of roentgenology</i> , Vol. xiv. (ed.)	554
<i>Rose and Carless' manual of surgery</i> (ed. C. P. G. Wakeley and J. B. Hunter)	547	Catterina, A. : <i>Die Bruchoperationen nach Bassini</i>	555
<i>The science and practice of surgery</i> (W. H. C. Romanis and P. H. Mittelner)	166	Collie, Sir J. : <i>Recent progress in medicine and surgery</i> (ed.)	549
<i>A short history of surgery</i> (Sir D'Arcy Power)	550	Cushing, H. : <i>Papers relating to the pituitary body, hypothalamus, and parasympathetic nervous system</i>	551
<i>Sixth international congress of military medicine and pharmacy, etc.</i> (W. S. Bainbridge)	169	Dorrance, G. M. : <i>The operative story of cleft palate</i>	717
<i>Die spezielle Chirurgie der Gehirnkrankheiten</i> (ed. F. Krause)	709	Davis, L. : <i>Intracranial tumors roentgenologically considered</i>	554
<i>Surgery of the stomach and duodenum</i> (J. S. Horsley)	713	Desjaques, et al. : <i>La phrénicectomie</i>	168
<i>The surgery of the sympathetic nervous system</i> (G. E. Gask and J. P. Ross)	719	Dumarest, F., et al. : <i>La phrénicectomie</i>	168
<i>Surgical anatomy</i> (C. L. Callander)	550	Egidi, G. : <i>Atlante di chirurgia dello stomaco</i>	378
<i>Surgical anatomy</i> (G. Massie)	716	Fevre, M. : <i>Chirurgie infantile d'urgence</i>	719
<i>Surgical operations : a text-book for students and nurses</i> (E. W. H. Groves)	168	Forrester-Brown, M., and Gordon, R. G. : <i>Paralysis in children</i>	715
		Foster, J., et al. : <i>The injured workman</i>	377
		Frontz, W. A., et al. : <i>History of urology</i>	552

	PAGE		PAGE
Reviews and Notices of Books, <i>contd.</i> —		Reviews and Notices of Books, <i>contd.</i> —	
Gabriel, W. B. : <i>The principles and practice of rectal surgery</i> - - -	161	Oehlecker : <i>Die Bluttransfusion</i> -	713
Gask, G. E., and Ross, J. P. : <i>The surgery of the sympathetic nervous system</i> - - -	719	Ogilvie, W. H. : <i>Treatment of fractures in general practice</i> - - -	377
Gordon, R. G., and Forrester-Brown, M. : <i>Paralysis in children</i> - - -	715	Perry, A., and Howard, R. : <i>The practice of surgery</i> - - -	518
Groves, E. W. H. : <i>Surgical operations : for students and nurses</i> -	168	Phelps W. M. and Kipluth R. J. H. : <i>The diagnosis and treatment of postural defects</i> - -	378
Guilheneuc, B.-O. : <i>De l'exclusion haute de l'estomac</i> - - -	164	Power, Sir D'Arcy : <i>A short history of surgery</i> - - -	550
Guleke, N., et al. : <i>Die spezielle Chirurgie der Gehirnkrankheiten</i> -	709	Ritson, S., et al. : <i>The injured workman</i> - - -	377
Hamer, H. G., et al. : <i>History of urology</i> - - -	552	Robson, J. H., et al. : <i>The injured workman</i> - - -	377
Harms, E., et al. : <i>Die spezielle Chirurgie der Gehirnkrankheiten</i> -	709	Romanis, W. H. C., and Mitchiner, P. H. : <i>The science and practice of surgery</i> - - -	166
Hartmann, H. : <i>Chirurgie du rectum</i> -	162	Ross, J. P., and Gask, G. E. : <i>The surgery of the sympathetic nervous system</i> - - -	719
Hertzler, A. E. : <i>The technic of local anaesthesia</i> - - -	553	Schum, H., and Krause, F. : <i>Die epileptischen Erkrankungen</i> -	709
Horsley, J. S. : <i>Surgery of the stomach and duodenum</i> - - -	713	Smart, M. : <i>The principles of treatment of muscles and joints by graduated muscular contractions</i> -	376
Howard, R., and Perry, A. : <i>The practice of surgery</i> - - -	548	Snidt, A., et al. : <i>Die spezielle Chirurgie der Gehirnkrankheiten</i> -	709
Hunter, J. B., and Wakeley, C. P. G. : <i>Rose and Carless' manual of surgery</i> (ed.) - - -	547	Smyth, jun., C. M., and Bickham, W. S. : <i>Operative surgery</i> - - -	518
Jorns, G., et al. : <i>Die spezielle Chirurgie der Gehirnkrankheiten</i> -	709	Souttar, H. S. : <i>The art of surgery</i> -	519
Jowett, R. E., et al. : <i>The injured workman</i> - - -	377	Stedman, T. L. : <i>A practical medical dictionary</i> - - -	716
Kaufmann, C. : <i>Handbuch der Unfallmedizin</i> - - -	555	Uffreduzzi, O. : <i>Trattato di patologia chirurgica generale e speciale</i> -	165
Keith, Sir A. : <i>Human embryology and morphology</i> - - -	710	Wakeley, C. P. G., and Hunter, J. B. : <i>Rose and Carless' manual of surgery</i> (ed.) - - -	547
Kellog, E. L. : <i>The duodenum</i> - - -	715	Walker, G. F., et al. : <i>The injured workman</i> - - -	377
Kemble, J. : <i>Idols and invalids</i> -	167	Williich, C. T., et al. : <i>Die spezielle Chirurgie der Gehirnkrankheiten</i> -	709
Kipluth, R. J. H., and Phelps, W. M. : <i>The diagnosis and treatment of postural defects</i> - - -	378	Rib approximator for use in lobectomy -	284
Krause, F., and Schum, H. : <i>Die epileptischen Erkrankungen</i> -	709	Richter's hernia - - -	601
Ledoux-Lebard, R. : <i>Manuel de radiodiagnostic clinique</i> - -	714	Rheumatism, disappearance of, in cancer - - -	627
Lewis, B., et al. : <i>History of urology</i> -	552	ROBERTS, J. F. H., and NELSON, H. P. : <i>Pulmonary lobectomy : technique and report of ten cases</i> -	277
Lewis, E. Catherine : <i>Urology in women</i> - - -	167	ROBERTS, R. E., and JONES, R. WATSON : <i>Calcification, decalcification, and ossification</i> - - -	461
Livingston, E. M. : <i>A clinical study of the abdominal cavity and peritoneum</i> - - -	169	Robertson-Lavalle operation for tuberculous joints - - -	475
Lower, W. E. : <i>Röntgenographic studies of the urinary system</i> -	379	ROBINSON, W. L. : <i>Bronchiectasis : a study of the pathology of sixteen surgical lobectomies for bronchiectasis</i> - - -	302
McAusland, S. : <i>The cure of hemorrhoids, varicose veins and ulceration, and allied conditions</i> - -	377	ROGERS, LAMBERT : <i>Associated facial and intracranial hamangiomata</i> -	229
Maddox, J. K. : <i>An introduction to 'overtin' rectal anaesthesia</i> -	163	Rona technique in blood lipase test in cancer - - -	627
Magnuson, P. B. : <i>Fractures</i> - - -	711	ROSS, J. PATTERSON : <i>Sympathectomy as an experiment in human physiology</i> - - -	5
Massie, G. : <i>Surgical anatomy</i> - - -	716		
Mitchiner, P. H., and Romanis, W. H. C. : <i>The science and practice of surgery</i> - - -	166		
Monro, T. K. : <i>The physician as a man of letters, science, and action</i> -	553		

SACRAL autonomic nerves, anatomy	100	670	Skull in hyperparathyroidism	22
Sacrocoecygeal region, sinuses in (see Coecygeal Sinus)			infectious at base, dislocation of atlas from	476
Sarco-carcinoma of liver	688		Slipped epiphysis of femur (see Femur)	
Sarcoma of bone, decalcification in	168		Slough, nasopharyngeal pouch in	250
— breast (left) 18 years after carcinoma of right	156		Smith (Edwin) Papayus	385
— kidney	306		Smith, N. Ross: Pothoculated hydatid cyst of liver	117
— liver, primary	687		Snares for use in lobectomy	264, 281
— metatarsal, diagnosis from march foot	131, 137, 142		'Snorting' test for competent palato-pharyngeal valve	355
— retroperitoneal, of left iliac region	612		Sodium antimony tartrate in biliary bladder	633
— with metastases	615		benzoate prior to prostatectomy	441
Scaphoid, carpal, fracture of, avascularity and	469		Soldiers, march foot in	133
— — — ununited	171, 482		Solitary plasmocytoma of the femur	373
— — — Preiser's disease of	482		Speech, cleft-palate, causes of	356
Scapula, sarcoma of, decalcification in	169		mechanism of	347
Schistosomiasis of bladder in England	632		training after operation for cleft palate	364
— hæmaturia due to	54		Spinal anaesthesia, effect on barium enema in megacolon	46
— cancer of liver and	689		in lobectomy	270, 279
Sclerosis and porosis of carpus, traumatic	482		cord, pressure on, in hypertonic dislocation of atlas	177
Sclerotics, blue, in hyperparathyroidism	22		from tumour of third cervical vertebra	516
Serotal hernia in mice under treatment with œstrin	507		Spine, benign giant-cell tumour of third cervical vertebra	513
Secretary, Editorial, presentation to	381		cervical, dislocation of, in ancient Egypt	386
Selenide treatment of cancer	619		Kummell's disease of, decalcification and	475
— — — case reports	624		metastases to, in renal cancer	389, 393
— — — duration of treatment in unfavourable cases	627		malignant adenoma of prostate: Pott's disease simulated	677
— — — prognosis	626		tuberculosis of, calcification of psoas abscess	161
— — — reaction in	625, 626		Splanchnic nerves, anatomy	669
— — — results	628		Splitting of fractures, decalcification and	467, 471, 482
— — — technique	624		Spontaneous dislocation of hip in childhood	523
— — — 'toilet operation' prior to	621		— fractures, calcification and	480
Semilunar, carpal, Kienböck's disease of cartilage (see Knee-joint)	483		— hyperemic dislocation of atlas	476
Seminal vesicles in genital tuberculosis	457		SSE treatment of cancer	619
Separation of epiphysis of femur (see Femur)			STEEL, W. AUKLEY: Mesenteric cyst as a cause of intestinal obstruction	104
Septum nasi, deviation of, in ancient Egypt	385		Stellate ganglion	86, 91, 92
Serum calcium in hyperparathyroidism	26, 28, 501		Stenosis of ileocecal valve	370
Sex, appendicitis mortality and	417		'Step-ladder' method of excising branchial fistula	180
— incidence of epidermoid, cysts of hands and fingers	34		Stomach, adenopapilloma of, with lymphoid hyperplasia of duodenum	519
— — — hæmaturia	46		— cancer of, recurrence twelve years after partial gastrectomy	542
— — — jejuno-ileal diverticula	187		Strangulated hernia	601
— — — osteo-chondritis dissecans	67		Strangulation of intestine (see Intestine, Acute Strangulation of)	
— — — primary carcinoma of liver	686		Stricture, urethral, hæmaturia with	54
— — — Raynaud's disease	613		Subperiosteal ossification	489
SMILES, N., and POLSON, CYRIL: A solitary plasmocytoma of the femur	373		Subphrenic abscess following lobectomy	287
Shock in pulmonary lobectomy	285		Suprapubic prostatectomy (see Prostatectomy)	
Sigmoid notch, hernia into, case report	158			
Silica, crystalline, injected into mesentery of rats' caecum: megacolon and	577			
SIMPSON, GRAHAM: Carcinoma of the kidney	388			
Sinus, coecygeal (see Coecygeal Sinus)				
— thrombosis in ancient Egypt	385			
Skull, fractures of, in ancient Egypt	2			

	PAGE		PAGE
Supraspinatus tendon, calcification of	466, 484	Tendons, fibromata of, calcification of	466
Surgical Cases, Some Early:—		Thoracic trunk sympathectomy, technique and results	122
The Edwin Smith Papyrus	1, 385	Thoracoplasty in bronchiectasis	260
Sutures in prostatectomy with closure	445	Thoracotomy v. laparotomy in diaphragmatic hernia of infants	430
Sweat, effect of sympathectomy on secretion of	13	Thorax in hyperparathyroidism	22
Sympathectomy	5, 113, 604	Thrombo-angiitis obliterans	605
— anatomy concerned	83, 94, 117	— etiology	606
— in angina pectoris	18	— medical treatment	608
— causalgia	12, 18, 116	— pathological picture	606
— effect on arteries	6, 9, 116, 604	— selection of cases for neurectomy	611
— arterioles and capillaries	11	— sympathectomy in	128, 609
— bladder	16	— results	612
— cardiac pain	17	— symptoms	606
— colon	15	— typhoid vaccine in	607
— pilomotor mechanism	13	Thrombosis, cerebral, in ancient Egypt	3
— muscular power	14	— lateral sinus, in ancient Egypt	385
— oculomotor phenomena	14	Tibia, acute osteomyelitis of, Winnett Orr treatment	530
— secretion of sweat	13	— and fibula, fractures of	474
— sensation	17	Tibial spine in etiology of osteo-chondritis dissecans	75
— as an experiment in human physiology	5	Tibialis anticus, fibroma of, calcification of	465
— hypogastric, for megacolon	129	Todd, A. T.: The selenide treatment of cancer	619
— lumbar	128, 609, 614	Tourniquets for use in lobectomy	264, 281
— methods of investigation	6	Toxæmia, acute strangulation and (see Intestine, Acute Strangulation of)	
— operations for ganglionectomy	8	Trachea and œsophagus, pouch due to imperfect separation of	244
— peri-arterial	115	Tracheo-œsophageal fistula, pouch associated with	243
— in Raynaud's disease	10, 120, 614	Trauma in etiology of march foot	133, 136
— temperature of affected part and	6, 9, 116, 604	— ossification in semilunar cartilage	400
— thoracic trunk, technique and results	122	— osteo-chondritis dissecans	74, 76, 78
— in thrombo-angiitis obliterans	128, 609, 612	— hamaturia due to	55
— vasomotor control	5, 604	— indirect, in etiology of slipped epiphysis of femur	318
Sympathetic nervous system (see also Nervous System)		Traumatic aneurysm of palmar arteries	215
— peripheral, anatomy of	83, 117	— cysts of hands and fingers (see Cysts of Hands and Fingers)	
— comparative	83	— sclerosis and porosis of carpus	482
— hypogastric nerves	96, 674	— subperiosteal hamatoma, ossification of	489
— innervation of bladder and ureter	96	Treitz, inferior duodenal fossa of	398
— distal part of colon	94	Trigonal flap, formation of, in prostatectomy with closure	446
— eye	86	Trumble, Hugh C.: The plan of the visceral nerves in the lumbar and sacral outflows of the autonomic nervous system	664
— lower extremity	102	Tuberculosis, genital, in male	456
— upper limb	88	— ascending infection	458
— rami and ganglia	90, 667	— classification of cases and results of treatment	458
— summary	102	— mortality	458
— pelvic plexus	100, 675	— radical operation	457
— trunks, anatomy	666	— orchidectomy in	456
— neurocytoma of	534	— summary	460
Syndesmitis metatarsia	133	— genito-urinary, hamaturia with	48, 52
Synovitis, dysenteric, spontaneous dislocation of hip following	523	— of hip, decalcification in	468
Syphilitic osteitis, bone changes in	481	— kidney	458
		— os calcis	462
TAYLOR, HERMON, and KEYNES, GEOFFREY: A case of parathyroid tumour	20		
Temperature of limb, effect of sympathectomy on	6, 9, 116, 604		
— records, sympathectomy and	6, 9		
Tendo Achillis, ossification of	466, 491		
Tendonitis ossificans	490		

	PAGE
Tuberculosis of spine, calcification of psoas abscess - - -	464
— — simulated by metastases from malignant adenoma of prostate	677
Tuberculous diverticulum of œsophagus — joints, Robertson-Lavalle operation	248
Twelfth nerve, embryonic cervical vestigial structures and - - -	475
239	
Typhoid vaccine in Buerger's disease -	607
ULCER, duodenal, repeated perforation of - - -	145
Ulnar artery, aneurysm of, in palm -	217
Ureter and bladder, innervation of -	96
Ureteric calculi, hæmaturia with -	52
— — and renal, of unusual size: repeated stone formation -	63
Urethra, stricture of, hæmaturia with -	54
— varicose veins of - - -	56
Urethral calculi, hæmaturia with -	52
Urethritis, hæmaturia with - - -	54
Urinary antiseptics prior to prostatectomy - - -	441
— tract, purpura of, hæmaturia with -	55, 61
— tuberculosis in male - - -	458
Urine, blood in (<i>see</i> Hæmaturia) — in genito-urinary tuberculosis -	458
— loss of calcium by, in hyperparathyroidism - - -	26, 27
Uroscleretan in diagnosis of carcinoma of kidney - - -	394
Urotropine prior to prostatectomy -	441
Uterus, fibroids of, calcification of -	466

VACCINE, typhoid, in Buerger's disease - - -	607
Varicose veins of bladder, hæmaturia from - - -	55
— — urethra - - -	56
Vas deferens, ligation of, prior to prostatectomy with closure - - -	441
— — tuberculosis of - - -	457
Vascular route in sympathetic innervation of upper limb - - -	88
Vasularity, relation to calcification of bone - - -	466, 468
Vaso-dilatation in causalgia - - -	12
Vasomotor control, sympathectomy and Veins, varicose, of bladder, hæmaturia from - - -	55
— — urethra - - -	56
Vertebra, third cervical, benign giant-cell tumour of - - -	513
Vesical (<i>see</i> Bladder)	
Vesicles, herpetic, formation of, in causalgia - - -	12
Vesico-vaginal fistula, hæmaturia with -	56
Vesiculectomy in genital tuberculosis -	457
Visceral motor mechanism, effect of sympathectomy on - - -	15
— sensation, effect of sympathectomy on -	17

WAKELEY, CECIL P. G.: A case of carcinoma of the right breast followed by sarcoma of the left breast after 18 years - - -	156
Walden's right mesenterico-parietal fossa - - -	398
WARD, R. OGLE, and CHRISTOPHERSON, J. B.: Billharzia disease in England: cystoscopic appearance of billharzia bladder before and after intravenous injections of sodium antimony tartrate -	632
WARDLE, W. E. M.: Cleft palate -	347
WARDLE, E. N.: Etiology and treatment of slipped epiphysis of the head of the femur - - -	313
Wax injection prior to operation on coccygeal sinus - - -	226
WEBB-JONSSON, A. E., and MEIN, E. G.: Adenopapilloma of stomach associated with lymphoid hyperplasia of duodenum - - -	519
Wengowski's theory of origin of bronchial fistula - - -	174, 182
Winnett Orr treatment of acute osteomyelitis of tibia - - -	530
— — — compound fractures - - -	474
Wiring the patella, Lister and - - -	557
WOOLLAND, H. H., and NORMAN, R. E.: The anatomy of the peripheral nervous system - - -	83
Wounds of head in ancient Egypt -	2
Wrist (<i>see</i> Carpal; Carpus)	

X-RAY appearances of bronchiectasis -	288
— — calcified hydatid cyst of omentum - - -	532
— — coccygeal sinus - - -	220, 226
— — diaphragmatic hernia in infants - - -	424, 428
— — gastric tumours - - -	521
— — march foot - - -	131, 141, 708
— — mesenteric cyst of ileum - - -	106
— — ossification of semilunar cartilage -	404
— — osteitis fibrosa with hyperparathyroidism - - -	22, 502
— — osteo-chondritis dissecans of ankle -	74
— — — hip and of pseudo-coxalgia -	73
— — — knee - - -	68
— — pseudo-coxalgia - - -	73
— — right paraduodenal hernia - - -	398
— — slipped femoral epiphysis - - -	322
— — spontaneous dislocation of hip -	524
— — thrombo-angiitis obliterans - - -	607
— — tumour of third cervical vertebra -	513
— aspect of jejuno-ileal diverticula -	199, 203
— diagnosis of diverticula of intestine -	335
— examination of branchial fistula - - -	175, 177, 179
— radiation and selenide treatment of cancer - - -	619
— significance of bone shadows - - -	467

ATLAS OF PATHOLOGICAL ANATOMY

ISSUED UNDER THE DIRECTION OF THE EDITORIAL COMMITTEE OF
The British Journal of Surgery.

FASCICULUS IX.
DISEASES OF THE GENITO-URINARY SYSTEM AND OF
THE APPENDIX

Compiled by E. K. MARTIN, M.S., F.R.C.S.

CONTENTS

	PAGE
TUBERCULOSIS OF THE KIDNEY - - - - -	285
TUBERCULOSIS OF KIDNEY - - - - -	287
A kidney divided by longitudinal section	
External surface (<i>coloured</i>) <i>p.</i> 288	
Section (<i>coloured</i>) <i>p.</i> 289	
TUBERCULOSIS OF KIDNEY - - - - -	290
A kidney divided by longitudinal section (<i>coloured</i>) <i>p.</i> 291	
TUBERCULOSIS OF KIDNEY - - - - -	292
One half of a left kidney, divided longitudinally (<i>coloured</i>) <i>p.</i> 293	
ACTINOMYCOSIS OF KIDNEY - - - - -	294
A kidney divided by longitudinal section (<i>coloured</i>) <i>p.</i> 295	
RUPTURE OF THE BLADDER - - - - -	296
RUPTURE OF BLADDER - - - - -	296
The contents of a male pelvis <i>p.</i> 297	
PAPILLOMA OF THE BLADDER - - - - -	298
PAPILLOMA OF BLADDER - - - - -	298
A bladder opened from the front <i>p.</i> 299	
DIVERTICULUM OF THE BLADDER - - - - -	300
DIVERTICULUM OF BLADDER. ENLARGED PROSTATE - - - - -	301
A bladder and prostate ; a portion of the anterior wall of the bladder has been removed <i>p.</i> 302	
SENILE ENLARGEMENT OF THE PROSTATE - - - - -	303
ENLARGED PROSTATE - - - - -	304
A bladder, prostate, and the greater part of the urethra opened from the front (<i>coloured</i>) <i>p.</i> 305	
CARCINOMA OF THE PROSTATE - - - - -	306
CARCINOMA OF PROSTATE - - - - -	307
The right kidney and ureter, the right half of the bladder and prostate, and part of the rectum <i>p.</i> 308	
ABSCESS OF THE PROSTATE - - - - -	309
RENAL HÆMORRHAGE FOLLOWING RAPID EMPTYING OF A DISTENDED BLADDER - - - - -	309
ABSCESS OF PROSTATE - - - - -	310
A bladder and prostate with parts of the penis and rectum <i>p.</i> 311	
RENAL HÆMORRHAGE FOLLOWING RAPID EMPTYING OF AN OBSTRUCTED BLADDER - - - - -	312
A bladder with the kidneys and ureters (<i>coloured</i>) <i>p.</i> 313	
STONE IN THE BLADDER - - - - -	314

	PAGE
CALCULI IN BLADDER AND KIDNEYS - - - - -	314
The kidneys, ureters, and bladder <i>p.</i> 315	
CARCINOMA OF THE BLADDER - - - - -	316
CARCINOMA OF BLADDER (ASSOCIATED WITH SIMPLE PAPILLOMA) - - -	316
A urinary bladder with a portion of the prostate <i>p.</i> 317	
HYDROCELE OF THE TUNICA VAGINALIS - - - - -	318
HYDROCELE OF TUNICA VAGINALIS - - - - -	318
A testicle with the sac of the tunica vaginalis distended into a hydrocele <i>p.</i> 319	
HÆMATOCHELE - - - - -	320
HÆMATOCHELE - - - - -	320
A portion of the spermatic cord, tunica vaginalis, and testis divided by longitudinal section (coloured) <i>p.</i> 321	
CYST OF THE EPIDIDYMIS - - - - -	322
CYST OF EPIDIDYMIS (SUPPURATION) - - - - -	322
A right testis, epididymis, and spermatic cord divided by vertical section (coloured) <i>p.</i> 323	
CYSTS OF EPIDIDYMIS (INFECTION) - - - - -	324
A multilocular cystic tumour divided by longitudinal section (coloured) <i>p.</i> 325	
TUMOURS OF THE TESTIS - - - - -	326
TERATOMA OF TESTIS (DERMOID CYST) - - - - -	327
A testicle opened from the front <i>p.</i> 327	
MIXED TUMOUR OF TESTIS - - - - -	328
A testis and epididymis divided by longitudinal section, together with a portion of the spermatic cord (coloured) <i>p.</i> 329	
CARCINOMA OF TESTIS - - - - -	330
One half of a testis with the spermatic cord (coloured) <i>p.</i> 331	
TORSION OF THE TESTIS - - - - -	332
TORSION OF TESTIS - - - - -	332
The left testis, epididymis, and spermatic cord (coloured) <i>p.</i> 332	
MIXED TUMOUR OF TESTIS - - - - -	333
A right testis divided by longitudinal section, with 5 in. of the spermatic cord <i>p.</i> 334	
CARCINOMA OF TESTIS - - - - -	335
A testis, tunica vaginalis, and portion of the spermatic cord, divided by longitudinal section (coloured) <i>p.</i> 336	
MIXED TUMOUR OF TESTIS - - - - -	337
The testis, together with a tumour in its vicinity (coloured) <i>p.</i> 338	
FIBROMYOMA OF SPERMATIC CORD - - - - -	339
A testis divided by longitudinal section, together with the spermatic cord (coloured) <i>p.</i> 340	
GUMMA OF THE TESTIS - - - - -	341
TUBERCULOSIS OF THE EPIDIDYMIS AND TESTIS - - - - -	341
GUMMA OF TESTIS - - - - -	342
A testis divided by longitudinal section, with part of the spermatic cord (coloured) <i>p.</i> 342	

	PAGE
GUMMA OF TESTIS - - - - -	343
One half of a testis divided by longitudinal section (<i>coloured</i>) p. 343	
TUBERCULOSIS OF EPIDIDYMS - - - - -	344
A left testis divided by vertical section (<i>coloured</i>) p. 344	
TUBERCULOSIS OF EPIDIDYMS - - - - -	345
A testis divided by longitudinal section, together with part of the spermatic cord (<i>coloured</i>) p. 345	
CARCINOMA OF THE PENIS - - - - -	346
CARCINOMA OF THE MALE URETHRA - - - - -	346
PAPILLOMA OF PENIS - - - - -	347
The anterior part of a penis removed after death p. 347	
CARCINOMA OF PENIS - - - - -	348
The anterior part of a penis divided by longitudinal section (<i>coloured</i>) p. 348	
CARCINOMA OF PENIS - - - - -	349
A glans penis from the front (<i>coloured</i>) p. 349	
CARCINOMA OF URETHRA - - - - -	350
The left half of a penis divided by longitudinal section p. 350	
MUCOCELE OF THE APPENDIX - - - - -	351
MUCOCELE OF APPENDIX - - - - -	352
A portion of the cæcum with the vermiform appendix (<i>coloured</i>) p. 353	
MUCOCELE OF APPENDIX (DIVERTICULA) - - - - -	354
The anterior half of a cæcum with part of the ascending colon and the appendix	
Surface (<i>coloured</i>) p. 355	
Section (<i>coloured</i>) p. 356	
MUCOCELE OF APPENDIX : PSEUDO-MYXOMA PERITONEI - - - - -	357
Parts of a vermiform appendix with a piece of omentum which was adherent to the larger fragment (<i>coloured</i>) p. 358	
ACUTE APPENDICITIS - - - - -	359
ACUTE APPENDICITIS - - - - -	361
A vermiform appendix removed by operation and divided by longitudinal section (<i>coloured</i>) p. 361	
ACUTE APPENDICITIS - - - - -	362
A vermiform appendix (<i>coloured</i>) p. 363	
SUBACUTE APPENDICITIS - - - - -	364
Part of a cæcum with a vermiform appendix divided by longitudinal section (<i>coloured</i>) p. 365	
PIGMENTATION OF APPENDIX - - - - -	366
A vermiform appendix divided longitudinally (<i>coloured</i>) p. 366	
INTUSSUSCEPTION OF APPENDIX - - - - -	367
A vermiform appendix with the adjacent part of the cæcum p. 367	
TORSION OF APPENDIX - - - - -	368
A vermiform appendix (<i>coloured</i>) p. 368	

L. TUBERCULOSIS OF THE KIDNEY.

TUBERCULOSIS of the kidney is a disease of both sexes, and is most common between the ages of 20 and 50. It is rare in childhood, and less common in old age than in early adult life. The infection tends to spread from the kidney down the ureter to the bladder, and in the male it frequently involves the genital tract as well. The tubercle bacillus is usually the only micro-organism present, and the disease is therefore often insidious in its onset and slow in its progress. If infection by pyogenic micro-organisms is superimposed on renal tuberculosis, the rate of destruction of the kidney is greatly increased.

The tubercle bacillus reaches the kidney through the blood-stream, into which intermittent showers of the micro-organisms may escape from any of the common sites of tuberculosis in lungs, lymphatic glands, or bones.

Tuberculosis of the kidney is unilateral at first, while a blood-borne infection must affect both kidneys to an equal extent. It is probable that minor lesions of both kidneys occur in patients who are the subjects of tuberculosis in any form, and that many of these relatively slight lesions heal, as in the case of tuberculosis elsewhere. But if tuberculosis of the kidney reaches a stage at which it becomes recognizable clinically, it usually progresses to complete destruction of the organ. Unless it can be checked by nephrectomy it spreads to the opposite kidney. The infection of the second kidney is probably hæmatogenous, though a spread along lymphatics is also possible.

It has been suggested that the tubercle bacilli may reach the kidney directly through the lymphatic vessels, either along the peri-ureteral lymphatics or by communications between the lymphatic systems of the lungs or alimentary canal and those of the kidney, but this hypothesis is opposed to clinical experience of urinary tuberculosis.

The initial lesion may appear either at the apex of a pyramid or in the medulla, and it is common to find several sites affected by the time the disease has reached the stage of clinical recognition. Tubercles are often present on the surface of the affected part of the kidney, and may be more easily recognized if the capsule is stripped. The tubercles coalesce and caseate, and if they are situated in the substance of the kidney a more or less complete fibrous capsule may be formed around them in an attempt, usually unsuccessful, to limit the spread of the disease.

Wherever the tubercles are deposited first, they tend when caseated to burst into the calices and to form ulcers which destroy progressively the pyramids and cortex of the kidney, so that the latter is converted into a hollow sac with a lobulated surface indicating the enlarged calices. The liquefied, necrotic material containing tubercle bacilli is discharged down the ureter so long as this remains patent.

While the hollowing out of the kidney is proceeding, tubercle bacilli spread along the lymphatic system of the calices, pelvis, and ureter. The mucous membrane becomes studded with tubercles, and is ultimately replaced by a tuberculous granulation tissue around which there is the usual thickening of the walls by chronic inflammatory fibrosis.

The enlargement of the kidney which accompanies its destruction is of moderate degree, and is seldom recognizable clinically unless a blocking of the lumen of the ureter has led to the formation of a tuberculous pyonephrosis or hydronephrosis. The kidney may even shrink if destruction of its parenchyma has been accompanied by much perinephric fibrosis. The perinephric fat is increased in quantity around a tuberculous kidney, and becomes tough and adherent to the kidney and to surrounding structures through fibrosis. It extends around the pelvis into the hilum. Tubercles and foci of caseation are seldom found in the perinephric tissue.

As the result of thickening of the wall of the ureter and of blocking of its lumen by caseous debris the affected kidney may be shut off from the rest of the urinary system. A kidney in this 'closed' type of renal tuberculosis is distended either with thick caseous material or with a watery fluid containing flakes of debris. A similar condition may arise locally in the kidney through blocking of the mouth of one calix. The affected calix with its corresponding renal parenchyma then becomes encysted in a fibrous capsule in which calcification is not uncommon.

The fibrosis of the ureter which accompanies renal tuberculosis may make the ureter thick enough to be felt on clinical examination. Contraction in the length of the ureter leads to a drawing up of the part of the bladder to which it is attached and contributes to the cystoscopic appearance of the ureteric orifice which is characteristic of renal tuberculosis. The first tubercles to appear in the bladder are situated behind the ureteric orifice, and tend to extend backwards before invading the trigone.

Actinomyces of the kidney is rare. The lesions produced are similar to those of tuberculosis.

TUBERCULOSIS OF KIDNEY.

A kidney divided by longitudinal section.

The specimen illustrates the results of advanced tuberculosis. The calices are greatly enlarged to form cavities lined by granulation tissue and filled with caseous material. The ureter is thickened and its lining mucous membrane has been replaced by necrotic granulation tissue. The external surface of the kidney is coarsely lobulated and is studded with tubercles.

Hunterian Museum, R.C.S., 853.1

CLINICAL HISTORY.—The patient was a boy, aged 12, who had been passing urine containing blood and pus. He recovered from the operation, but returned to hospital and died nineteen months later.

AUTOPSY.—Tuberculosis of remaining kidney and ureter, bladder, seminal vesicles, and both lungs.

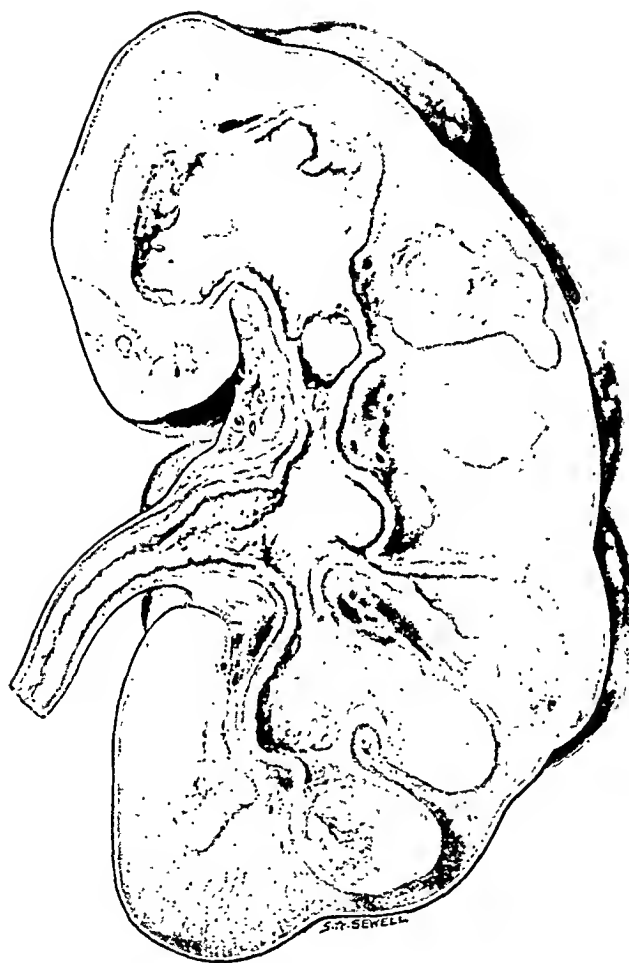
TUBERCULOSIS OF KIDNEY.



EXTERNAL SURFACE.

HUNTERIAN MUSEUM, R.C.S., 853.1

TUBERCULOSIS OF KIDNEY.



SECTION.

HUNTERIAN MUSEUM, R.C.S., 853.1

NO. 33—SUPPLEMENT

M 1

TUBERCULOSIS OF KIDNEY.

A kidney divided by longitudinal section.

At the upper pole a group of tubercles is situated in the cortex. Near these there is a small cavity due to a softening of an older tuberculous focus. This cavity opens into one of the calices. There are a number of tubercles in the mucous membrane of the pelvis and calices.

Hunterian Museum, R.C.S., 834.1

CLINICAL HISTORY.—The patient was a woman, aged 20, who had suffered from pain which extended from the region of the right kidney down to the bladder. There was also pain over the buttock and down the inner side of the thigh. Micturition was frequent and painful.

On examination tubercle bacilli were found in the urine, which contained a moderate quantity of pus. Cystoscopy showed ulceration of the right ureteric orifice. There was a patch of inflamed mucosa studded with tubercles to the right of and above the right ureteric orifice and another higher up and to the left. The left ureteric orifice was normal and the efflux clear.

The right kidney was removed and the vesical irritation gradually subsided. The tubercle bacilli eventually disappeared from the urine.

For the next two and a half years the patient was apparently in perfect health and was able to take active exercise. Her health then began to fail, tubercle bacilli were found in the urine, and vesical symptoms returned. She died with uræmic symptoms three years and ten months after the nephrectomy.

TUBERCULOSIS OF KIDNEY.



HUNTERIAN MUSEUM, R.C.S., 834.1

TUBERCULOSIS OF KIDNEY.

One half of a left kidney, divided longitudinally.

The kidney is enlarged and lobulated, but the amount of renal substance is small. The increase in size is due to dilatation of the pelvis and calices caused by obstruction of the ureter. The calices are lined by a shaggy, purulent material, and the ureter is almost closed by inflammatory thickening of its wall.

Museum of St. Bartholomew's Hospital, Q.81

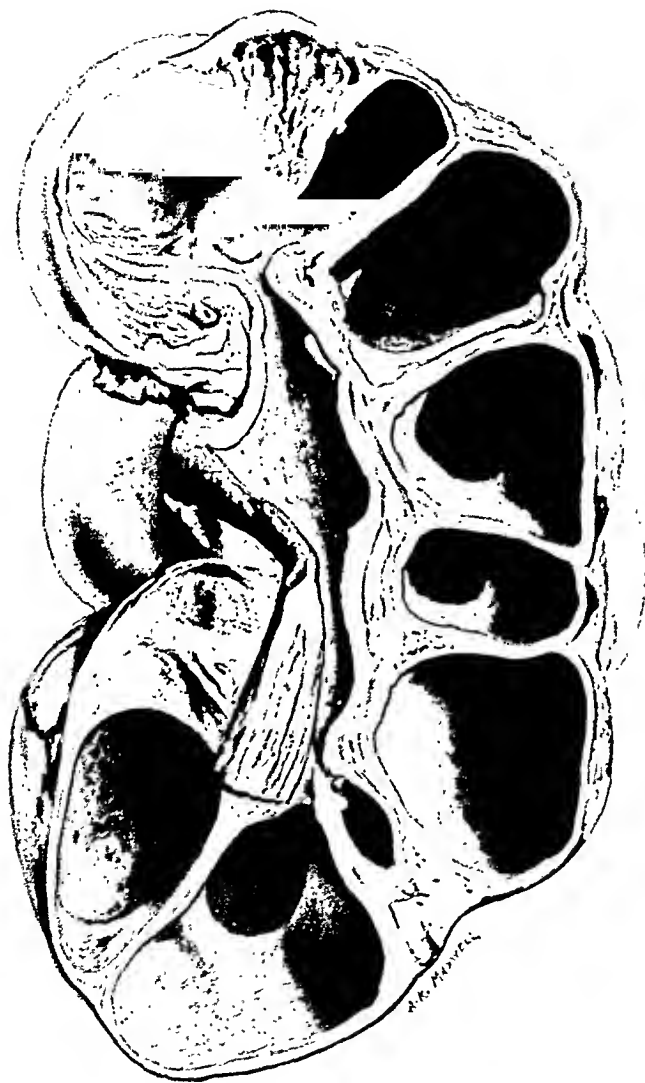
MICROSCOPIC STRUCTURE.—The appearances are characteristic of fibro-cascous tuberculosis.

CLINICAL HISTORY.—The patient was a man, aged 25, who for twelve months had had frequency of micturition every half-hour by day and four or five times by night. At the beginning, he had had an attack of hæmaturia, and he had passed clots of blood at intervals throughout his illness.

The urine was acid and contained pus, but no tubercle bacilli were found. The kidney could not be felt. The diagnosis was made by cystoscopy.

Removal of the kidney was followed by uninterrupted recovery.

TUBERCULOSIS OF KIDNEY.



MUSEUM OF ST. BARTHOLOMEW'S HOSPITAL, Q.81

ACTINOMYCOSIS OF KIDNEY.

A kidney divided by longitudinal section.

The kidney is enlarged and its external surface is lobulated. The surface of the section shows that the substance of the kidney has been almost entirely destroyed and that the greatly distended calices are lined by granulation tissue.

Hunterian Museum, R.C.S., 946.1

MICROSCOPIC STRUCTURE.—Actinomycosis.

CLINICAL HISTORY.—The patient was a married woman, aged 31, who had suffered from severe pain in the right loin for two years before admission to hospital. The pain disappeared, but during a pregnancy which terminated six weeks before admission she had not been feeling well and had lost flesh. At the confinement a swelling was noticed on the right side of the abdomen. Shortly afterwards she had three rigors in succession.

On admission to hospital her temperature was 102°, and there was a large firm swelling which involved the right iliac, right hypochondriac, and umbilical regions. It moved slightly with respiration and was not tender. The urine was acid, s.g. 1027, and contained no albumin or blood. Three days later her temperature rose to 104° and there was profuse sweating. She made a good recovery after nephrectomy.

AFTER-HISTORY.—Eight months later there were râles and bronchial breathing at the apex of the left lung.

ACTINOMYCOSIS OF KIDNEY.



HUNTERIAN MUSEUM, R.C.S., 946.1

LI. RUPTURE OF THE BLADDER.

RUPTURE of the bladder is commonly due either to a blow on the abdomen when the bladder is distended or to an injury associated with fracture of the pelvis. In the first case the patient is intoxicated, the rupture is usually intraperitoneal, and the urine escapes into the peritoneal cavity. In the second case the rupture is extraperitoneal, and is caused either by penetration of the bladder by one of the fragments of bone or by tearing of the bladder at or near its junction with the urethra. The urine escapes into the cellular tissue of the pelvis.

RUPTURE OF BLADDER.

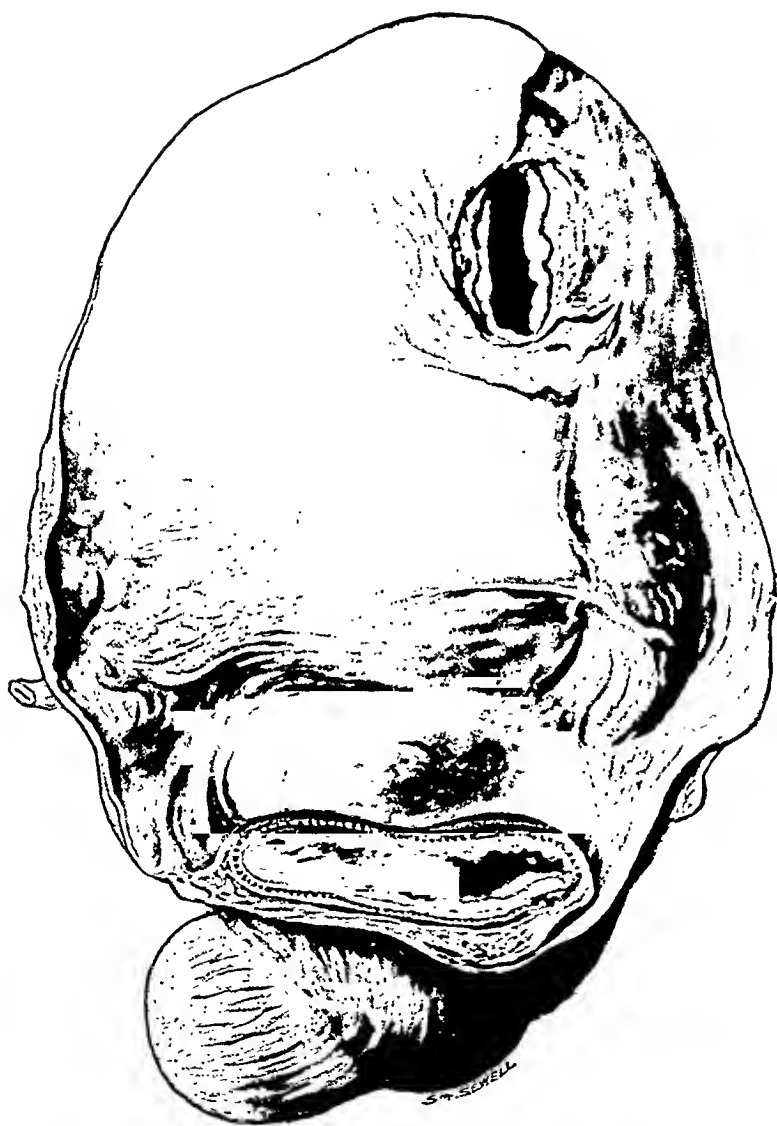
The contents of a male pelvis.

There is an intraperitoneal rupture of the bladder slightly to the right of the middle line and just behind the apex. It has the form of a vertical slit $\frac{3}{4}$ in. long. The walls of the bladder appear healthy. A layer of inflammatory exudate lines the rectovesical pouch.

Hunterian Museum, R.C.S., 7490.1

CLINICAL HISTORY.—The patient was a man, aged 25, who while intoxicated fell from the box seat of a wagon, two of the wheels of which passed over his abdomen. On admission to hospital he had abdominal pain and vomiting, and blood-stained urine was drawn off by a catheter. After this he rapidly developed tenderness and distension of the abdomen and dullness of the flanks, especially on the right side. He died on the second day after the injury.

RUPTURE OF BLADDER.



HUNTERIAN MUSEUM, R.C.S., 7490.1

LII. PAPILLOMA OF THE BLADDER.

PAPILLOMA is the only common simple tumour of the bladder, and, like similar growths elsewhere, shows a definite tendency towards malignancy. In nearly half the cases the papillomata are multiple when first seen. They are most common between the ages of 40 and 60.

A papilloma of the bladder is composed of a branching core of vascular fibrous tissue which may contain unstriped muscle fibres and is covered by transitional epithelium. The pedicle may be narrow or broad, and the papillae which branch from it are characteristically long and thin, but may be represented merely by lobulations of the surface.

The usual site of papillomata is the neighbourhood of a ureteric orifice or around the edge of the trigone, though they may spread over any part of the bladder. They have a strong tendency to increase in number, to recur after removal, and to end in carcinoma. Gross ulceration and incrustation with phosphates is not common. A papilloma of the bladder bleeds readily because of its great vascularity, and, if pedunculated, may obstruct the internal meatus or the orifice of a ureter.

PAPILLOMA OF BLADDER.

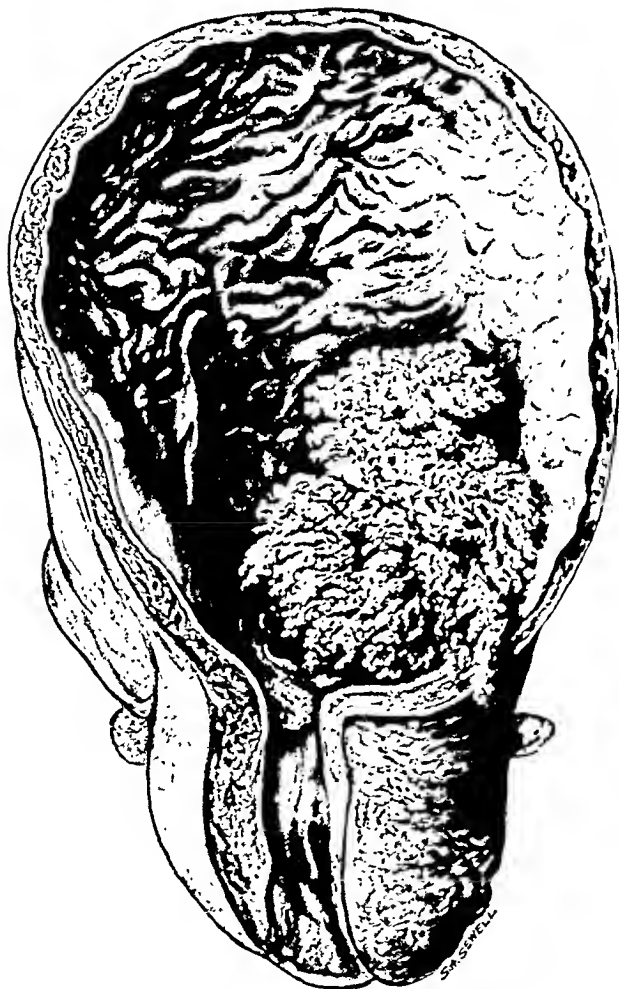
A bladder opened from the front.

A villous papilloma $1\frac{1}{2}$ in. in diameter has grown from the mucous membrane in the neighbourhood of the left ureteric orifice. The primary processes of the tumour are thickly beset with finer secondary villi. The interior of the bladder is fasciculated.

Hunterian Museum, R.C.S., 1594.1

CLINICAL HISTORY.—The patient was an old man who had suffered for over eight years from hæmaturia.

PAPILLOMA OF BLADDER.



HUNTERIAN MUSEUM, R.C.S., 1504.1

LIII. DIVERTICULUM OF THE BLADDER.

IN the presence of long-continued obstruction to the emptying of the bladder small sacculi are often formed by protrusion of the mucous membrane between the muscle bundles. Under similar conditions one or more large diverticula may be formed, and no valid distinction can be drawn between these and the sacculi found in cases of prostatic obstruction. Diverticula also occur in the absence of demonstrable obstruction to the outflow from the bladder, and an attempt has been made to limit the term 'diverticulum' to this variety, a congenital weakness of the bladder wall being postulated to account for its development.

A diverticulum is composed of a lining of mucous membrane covered by a layer of fibrous tissue in which there may be a small amount of unstripped muscle. It may be of any size up to that of the bladder itself, and is not necessarily single. It communicates with the bladder by a narrow aperture which may be situated in any part of the viscus, but is usually behind and lateral to the ureteric orifice. The edge of the opening is well defined and the neighbouring part of the bladder wall is usually trabeculated, even though the remainder has a normal appearance.

As a diverticulum grows in size its communication with the bladder may enlarge or may remain constant. If it is situated near the ureteric orifice and the diverticulum increases in size, the ureteric orifice may be drawn into the pouch. A large diverticulum may obstruct the lower end of the ureter by pressure, or may interfere with micturition by distortion of the normal relations of the bladder. It is apt to become infected owing to the absence of an effective muscular coat and a consequent inability to empty itself. A calculus may be formed within it as a result of infection. Carcinoma may develop at or near the mouth of a diverticulum.

DIVERTICULUM OF BLADDER. ENLARGED PROSTATE.

A bladder and prostate; a portion of the anterior wall of the bladder has been removed.

The prostate is generally enlarged, and from its upper surface a trilobed mass projects into the cavity of the bladder and obstructs the urethral orifice. The bladder is greatly dilated and hypertrophied and its inner surface is fascieulated. There is a sacculus 3 in. in diameter to the right side and above the right ureteric orifice. Both ureters are dilated and the vesiculae are atrophied.

Hunterian Museum, R.C.S., 7675.1

CLINICAL HISTORY.—The patient was a man, aged 75, who died from a cut throat self-inflicted during a fit of depression.

AUTOPSY.—The bladder was distended to the umbilicus. There was a dilatation of the pelvis of the kidney on both sides but the calices were not much affected. There was no evidence of pyelonephritis.

ENLARGED PROSTATE.

A bladder, prostate, and the greater part of the urethra opened from the front.

The prostate is enlarged and its middle lobe projects upwards into the bladder. The nodular nature of the enlargement can be seen on the cut surface of the gland. The muscular wall of the bladder is much hypertrophied and its inflamed mucous membrane is covered here and there by exudate.

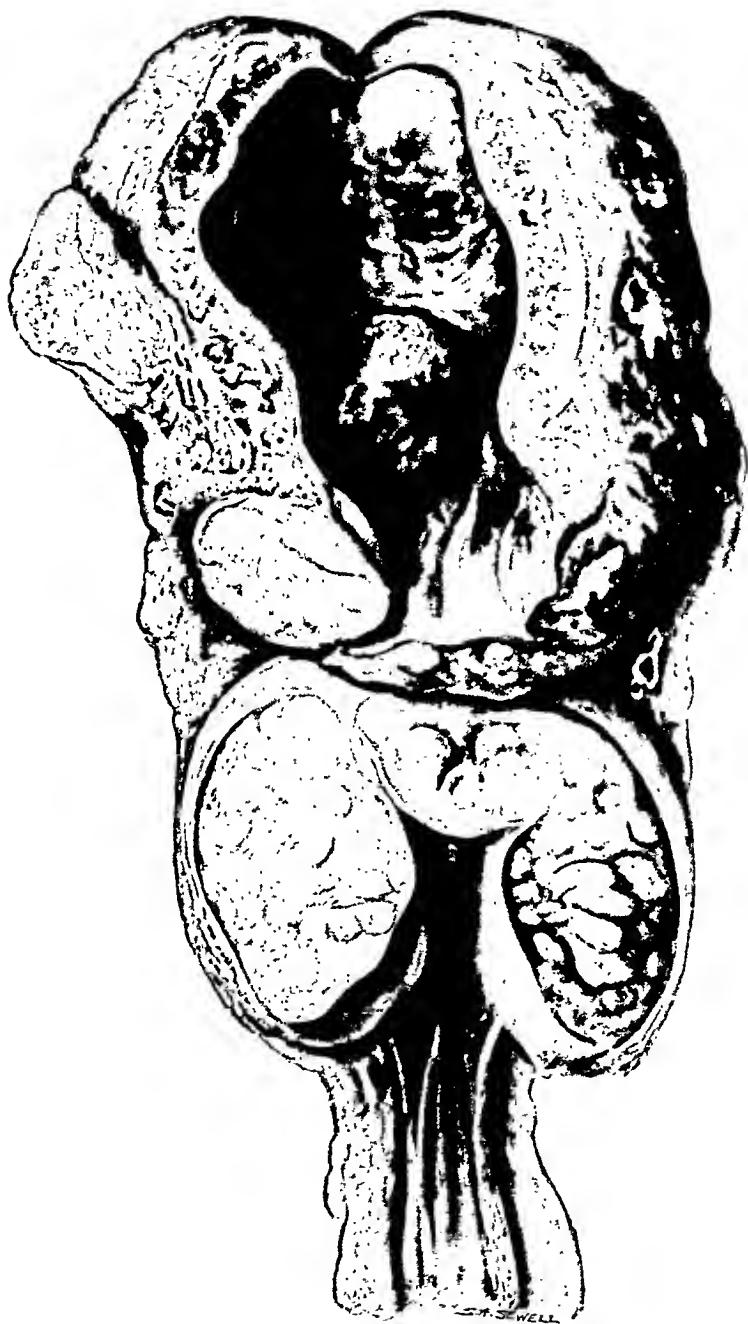
Museum of University College Hospital, 3BL.18

CLINICAL HISTORY.—The patient was a man, aged 76, who had complained of difficulty in passing water for four years, during which he had been catheterized at irregular intervals. For the ten days before admission he had had complete retention, which was relieved by catheterization.

On admission the bladder was distended to the umbilicus and the tongue was dry. On rectal examination a large soft prostate was felt. Suprapubic cystostomy was performed under local anæsthesia. The bladder contained blood-stained urine and a stone about 2 in. in diameter. Five days later the blood-urea was 332 mgrm. per cent. The patient died of bronchopneumonia nine days after admission.

AUTOPSY.—The left kidney was almost completely atrophied and its ureter was only about as thick as a thread. The right ureter and pelvis were dilated and contained pus, and there were numerous abscesses in the right kidney.

ENLARGED PROSTATE.



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 3BL.18

LV. CARCINOMA OF THE PROSTATE.

CARCINOMA of the prostate most often begins between the ages of 60 and 70, and has no recognized connection with senile enlargement or with prostatitis. Microscopically the growth is usually of scirrhus type, but may also be an adenocarcinoma with cubical or columnar cells.

In the common scirrhus form the prostate is enlarged slightly or not at all, but becomes hard and nodular and the posterior median groove is lost. The gland becomes fixed to surrounding structures by infiltration. Owing to the freedom of their lymphatic communication the first extension of a carcinoma of the prostate is into the vesiculæ seminales, the lymph-glands, and the cellular tissue of the pelvis. In the bladder the growth appears first in the form of submucous nodules which ulcerate later. Ulceration into the prostatic urethra is common in the later stages. Posteriorly the growth spreads backwards in the pelvic cellular tissue on either side of the rectum and may diminish its lumen. One or both ureters may become obstructed, and nodules of growth may appear on the peritoneum of the recto-vesical pouch.

In adeno-carcinoma the prostate is enlarged and nodular, but may be soft enough to be mistaken for a non-malignant enlargement of the senile type.

Carcinoma of the prostate is apt to cause skeletal metastases which are of osteoplastic type. The new bone formed is usually spongy and abundant. Visceral metastases are uncommon.

Sarcoma of the prostate is rare.

CARCINOMA OF PROSTATE.

The right kidney and ureter, the right half of the bladder and prostate, and part of the rectum.

The cut surface of the specimen shows that the position of the prostate is occupied by a fibrous growth in the meshes of which numerous minute opaque, yellow granules are visible. No normal prostatic tissue can be seen. Above and below the urethra the growth extends anteriorly beyond the limits of the specimen. Upwards the tumour extends into the base of the bladder, and fungating masses are present both in front of and behind the internal meatus, the largest being behind. Below the base of the bladder the growth extends round the vesicula seminalis, and from this extension a process of growth can be seen spreading subperitoneally outside the posterior wall of the bladder. Numerous subperitoneal nodules of growth are present on both surfaces of the recto-vesical pouch. The bladder wall is hypertrophied and the mucous membrane congested and ulcerated in places. The wall of the rectum is not invaded by growth. Behind the rectum the cut surfaces of two lymphatic glands containing growth can be seen.

The kidney is not enlarged externally. A cyst the size of a bean is present on its convex border just below the middle. The external surface is smooth. The cut surface shows that the pelvis is considerably dilated and the papillæ have been completely absorbed, leading to dilatation of the calices. The lining membrane is smooth and not congested. The cortex is diminished in thickness. The renal artery is thickened.

The ureter is dilated in its upper two-thirds. A vertical section has been made on the postero-external aspect of the specimen, opening the lumen of the ureter below the level of the pelvic brim. The cut surface shows the whole pelvic cellular tissue infiltrated with growth which surrounds the vessels and ureter. The growth has invaded the wall of the ureter and grown into its lumen, completely obstructing it shortly before its entry into the bladder. The external iliac vein is thrombosed.

Museum of University College Hospital, 6BL.11

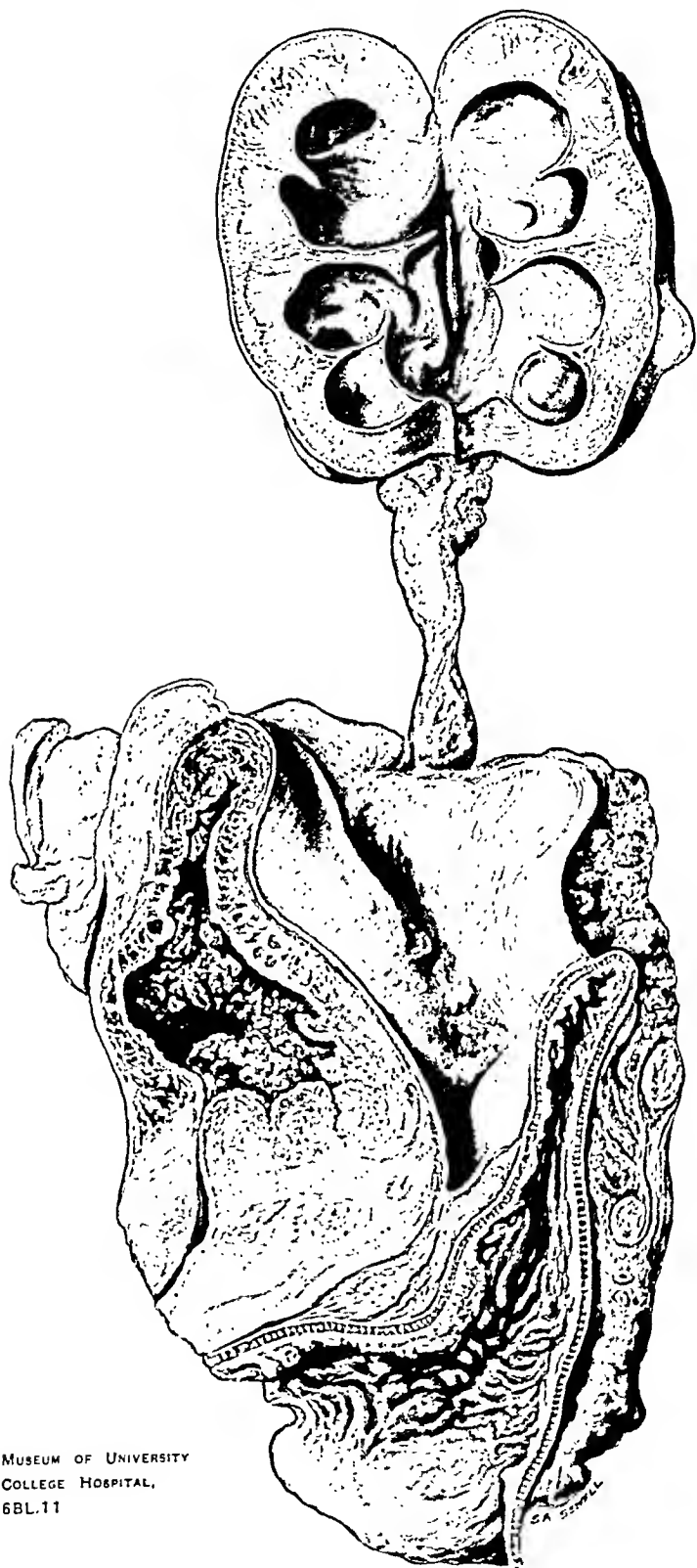
MICROSCOPIC STRUCTURE.—Carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 62. On admission he complained of swelling of the right leg for one to two weeks, hæmaturia for one month, difficulty in micturition for two weeks, frequency of micturition and pain across the lower lumbar region for two weeks. For two years he had had bleeding from the rectum, and five months before admission an attack of this lasted about two months and was associated with pain across the lower part of the abdomen.

On admission the urine was red with blood, there was œdema of the whole right lower limb, and on rectal examination a hard fixed mass was present in the position of the prostate extending up out of reach. Four days after admission a suprapubic cystotomy was performed to relieve retention due to blood-clots in the bladder. The patient became drowsy on the fourth day after the operation and died on the ninth day.

AUTOPSY.—Small growths were present in the lungs and liver, the right internal iliac vein was filled with growth, the left ureter was not dilated, and the growth in the pelvic cellular tissue extended upwards in front of the lumbar spine.

CARCINOMA OF PROSTATE.



MUSEUM OF UNIVERSITY
COLLEGE HOSPITAL,
6BL.11

LVI. ABSCESS OF THE PROSTATE.

ACUTE suppuration in the prostate is usually a sequel of gonorrhoea or of urethritis caused by the passage of instruments.

Numerous small abscesses, or, by coalescence of these, one large abscess, may be formed in the prostate. If not opened, the abscesses burst into the urethra. Less commonly a prostatic abscess may discharge into the rectum or on to the surface through the perineum.

LVII. RENAL HÆMORRHAGE FOLLOWING RAPID EMPTYING OF A DISTENDED BLADDER.

IN the presence of long-continued obstruction to the outflow of urine from the bladder the renal circulation becomes adapted to a condition of constantly raised pressure in the pelvis and calices. If this balance becomes disturbed by the sudden emptying of the distended bladder, as by the passage of a catheter, the kidneys may be fatally disorganized by hæmorrhage into their substance and into the pelvis.

ABSCCESS OF PROSTATE.

A bladder and prostate with parts of the penis and rectum.

The prostate is riddled with abscess cavities; the prostatic urethra is extensively ulcerated and communicates with the abscesses by large apertures. The bladder wall is hypertrophied and the mucous membrane shaggy from inflammation. There is an abscess cavity between the prostate and the rectum.

Hunterian Museum, R.C.S., 7692.1

MICROSCOPIC STRUCTURE.—Dense leucocytic infiltration of prostate. No evidence of tuberculosis.

No clinical history.

AUTOPSY.—"By desire of Mr. Hayes and in the presence of him and Mr. Moffat, I opened the body of a young man. I took out the penis, bladder, and rectum; I then opened the bladder on the fore part and found it very thick in its muscular coat, of a livid, pale colour. The inner surface was shaggy, especially at the opening of the ureters, which were very large in their whole length; so were the pelvis and kidneys. All these cavities were filled with a pale brown mucus. I then slit open the urethra through its whole length. On continuing the incision from the fore part of the bladder through the prostate gland and body of the penis, first we found in the body of the prostate gland and membranous part a vast number of irregular cavities which seemed to have been the seat of abscesses which had burst into the urethra so that here the urethra was very irregular or wanting in a great many places.

"There had one abscess formed in the posterior part of the prostate which had destroyed the termination of the ducts and testicles and that part of the vesiculae seminales which is next to the gland, so that the communication between these parts and the urethra was cut off, and their communication with one another. I opened the vesiculae seminales and observed that their contents were the same as that which we find in common in dead bodies, and he had not been dead above 11 hours, so that no putrefaction could have taken place. The contents of the vasa deferentia and the contents of these bags had a free passage into this abscess on the inside of the urethra. About the bulbous part there was a loss of substance, which might be called an ulcer. The urethra was sound from this to the glans, excepting where the mortification had communicated with it.

"Why should there be some disease in the kidneys? For they discharged a mucus. These parts seem all to have some consent with one another."

(Hunterian MS.—'Dissections of Morbid Bodies', No. 125, p. 212.)

ABSCESS OF PROSTATE.



HUNTERIAN MUSEUM, R.C.S., 7692.1

RENAL HÆMORRHAGE FOLLOWING RAPID EMPTYING OF AN OBSTRUCTED BLADDER.

A bladder with the kidneys and ureters.

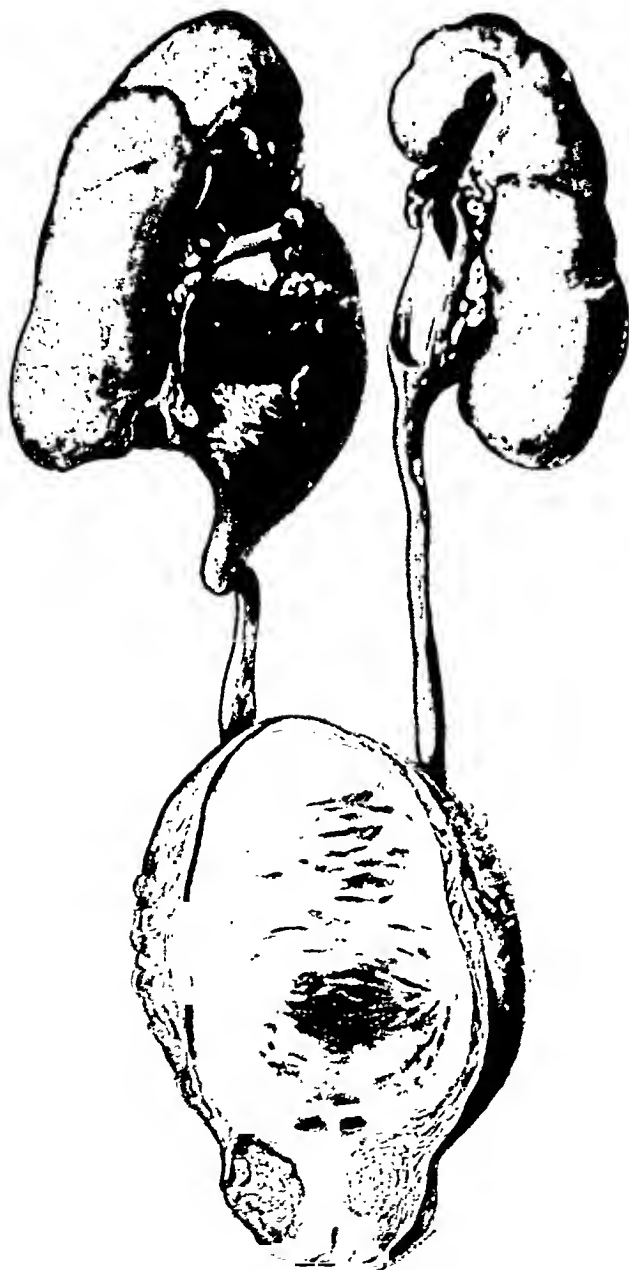
The bladder has been opened in front. It is dilated and trabeculated owing to an enlargement of the prostate. The right kidney is dilated and its pelvis and calices are distended with blood. The right ureter is dilated and distended with blood. The left kidney is in the same condition but to a lesser degree.

MICROSCOPIC STRUCTURE.—The kidney substance is permeated by a uniform diffuse actively growing fibrous tissue. In a few places there is lymphocytic infiltration. The capsules of the glomeruli are vacant and the glomeruli are all rather swollen. The tubular epithelium is swollen and irregular and stains badly. A few tubules contain blood. The arterioles show moderate proliferation of the intima.

Museum of King's College Hospital, 0102B

CLINICAL HISTORY.—The patient was a man, aged 60, who had had digestive disturbance for many months but never called his doctor's attention to any urinary trouble. While under treatment for indigestion he developed acute retention of urine, and his bladder, which extended to the umbilicus, was rapidly emptied by a catheter. Vomiting, hiccups, and a rigor followed at a short interval. He was admitted to hospital in a state of uræmia, and died four days after the passage of the catheter.

RENAL HÆMORRHAGE FOLLOWING RAPID EMPTYING OF AN
OBSTRUCTED BLADDER.



MUSEUM OF KING'S COLLEGE HOSPITAL, 0102B

NO. 34—SUPPLEMENT

LVIII. STONE IN THE BLADDER.

CALCULI may reach the bladder from the kidney and be retained, or they may be formed in the bladder. Those which are formed in the kidney and retained in the bladder consist usually of uric acid or calcium oxalate, and they may increase slowly in size without leading to infection of the bladder. If the bladder becomes infected, the stone is likely to grow rapidly by deposition of calcium and ammonio-magnesium phosphates on its surface.

Phosphatic stones are liable to be formed in the bladder when obstruction to the outflow of urine is combined with infection. They are therefore common in cases of enlargement of the prostate with cystitis, and in diverticula. Phosphatic stones also form readily on any foreign body in the bladder. Occasionally the bladder becomes tightly contracted round a large stone.

Pyelonephritis is the usual cause of death in unrelieved cases of vesical calculus.

CALCULI IN BLADDER AND KIDNEYS.

The kidneys, ureters, and bladder.

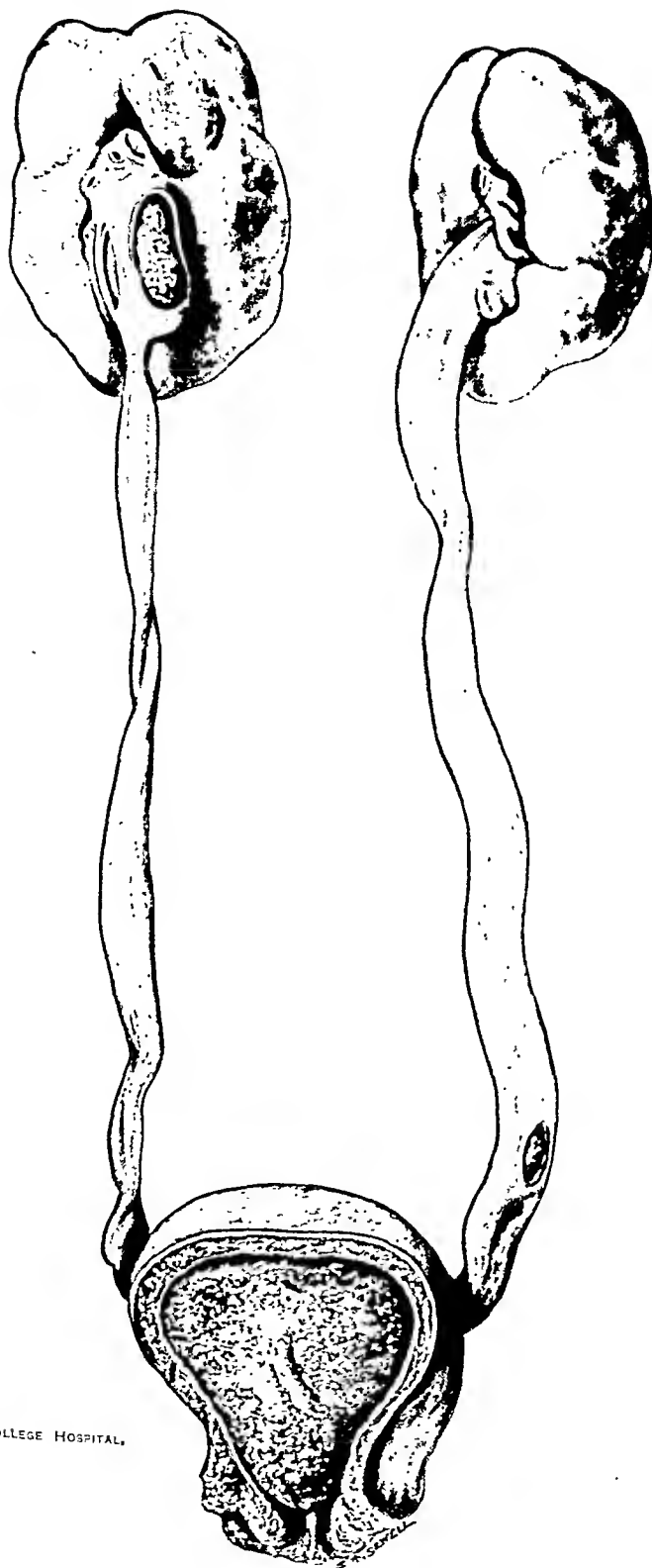
In the right kidney the dilated pelvis is completely filled by a uric acid calculus thinly covered with phosphates. The calices are moderately dilated and in the highest is another small calculus. The right ureter is slightly dilated and thickened.

In the left kidney the pelvis and calices are dilated and at both extremities the renal substance is thinned. The left ureter is dilated and contains two small stones immediately above its entrance into the bladder. Another stone blocks its lumen at a higher level.

The bladder is contracted and hypertrophied. Its cavity is completely filled by a large calculus with an irregularly pyramidal outline, the smaller extremity occupying the neck of the bladder. The stone is composed of two smaller stones feebly united. Each part presents a fawn-coloured nucleus of uric acid surrounded by concentric laminae and covered superficially by a rough, greyish-white layer of phosphates.

Museum of University College Hospital, 11.BF.9

CLINICAL HISTORY.—The patient was a man who was the subject of heart disease. No operation was performed.



MUSEUM OF
UNIVERSITY COLLEGE HOSPITAL,
11.BF.9

LIX. CARCINOMA OF THE BLADDER.

PRI-MARY carcinoma of the bladder is almost always of the papillary type. The papilloma which represents the first stage appreciable clinically may be malignant from the first or may become malignant after a long period of slow growth during which it appears to be benign.

Invasion of the pedicle of a papilloma and of the subjacent part of the bladder wall by epithelium is a positive proof of malignant change, and irregularity of size and type of the epithelial cells covering the papilloma is usually present in one which has become malignant.

Glandular carcinoma with mucinous degeneration of its columnar cells occasionally arises in the tubular glands around the neck of the bladder. It tends to infiltrate the wall diffusely.

CARCINOMA OF BLADDER.

(ASSOCIATED WITH SIMPLE PAPILLOMA.)

A urinary bladder with a portion of the prostate.

The inner surface of the bladder, except over the trigone and the lower part of the posterior wall, is raised by a diffuse and ragged growth which has invaded deeply into the wall.

Within the concavity of the upper border of the growth is a simple papilloma, attached to the mucous membrane by a narrow pedicle and wholly unconnected with the malignant disease. The villous surface which the latter presents indicates that it arose as a papilloma, at first probably benign in character.

The dilated ureters are indicated by rods.

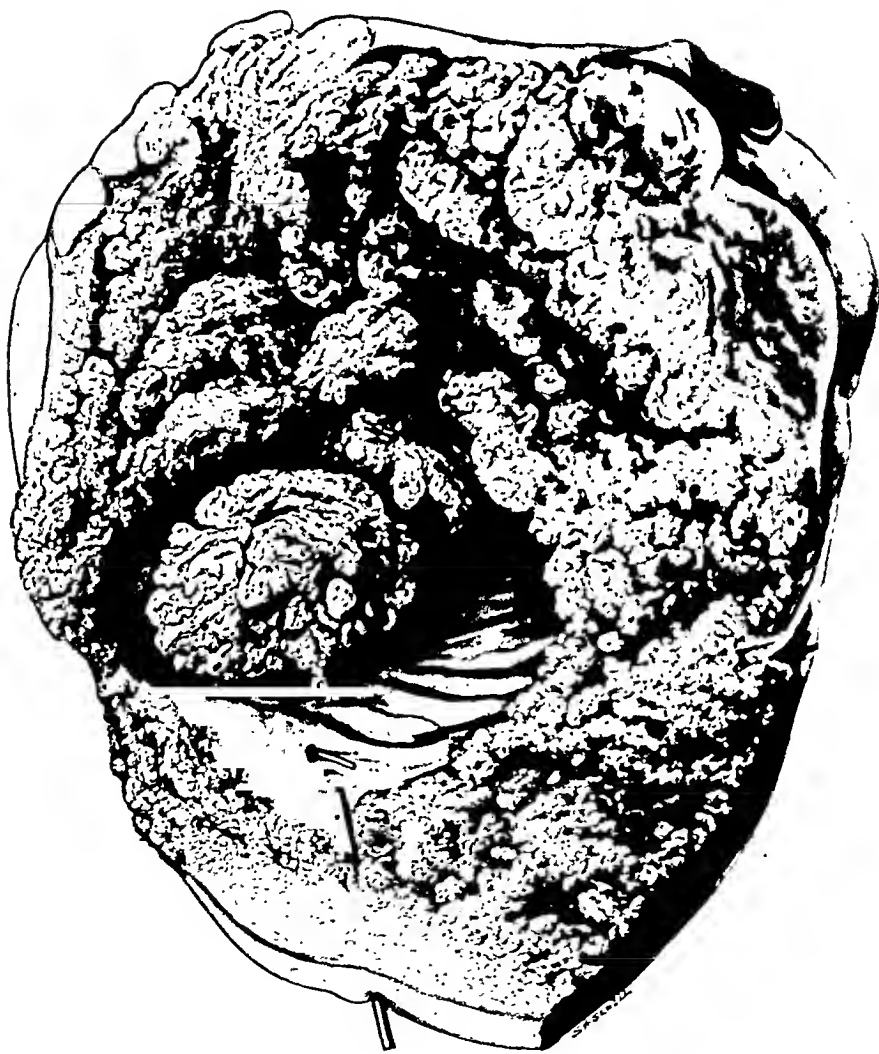
Hunterian Museum, R.C.S., 2144.1

MICROSCOPIC STRUCTURE.—Transitional-celled carcinoma. In the deeper parts of the growth the central elements of the cell-columnus are large and flat, and here and there small cell-nests have been formed.

CLINICAL HISTORY.—The patient was a man, aged 58, who was admitted to hospital on account of pain and difficulty in micturition. His illness began three years before with attacks of hæmaturia. During the last nine months of life there was severe hæmorrhage from the bladder, painful and frequent micturition, and loss of flesh.

AUTOPSY.—Bladder enlarged, with omentum and intestine adherent to its posterior surface. Suppuration between anterior abdominal wall and bladder. Bilateral pyelonephritis.

CARCINOMA OF BLADDER.
(ASSOCIATED WITH SIMPLE PAPILLOMA.)



HUNTERIAN MUSEUM, R.C.S., 2144.1

LXI. HÆMATOCELE.

A HÆMATOCELE is formed when the tunica vaginalis is distended with blood. It is usually, but not necessarily, the result of hæmorrhage into a pre-existing hydrocele.

The hæmorrhage may occur spontaneously, but is more often the result of injury, such as the tapping of a hydrocele. It may also follow a blow or any severe muscular exertion. In some cases a hæmatocele grows slowly and discontinuously by repeated hæmorrhages. Suppuration is uncommon unless infection has been introduced during tapping.

On section of a recent hæmatocele the cavity of the tunica is filled with fluid or clotted blood and there is usually some extravasation into the scrotum. On section of a hæmatocele which has been present for some time the tunica is thickened and lined by organized clot. The cavity is occupied by a brownish fluid which may contain crystals of cholesterol. The tunica albuginea may be thickened and the testis may be compressed and fibrotic.

HÆMATOCELE.

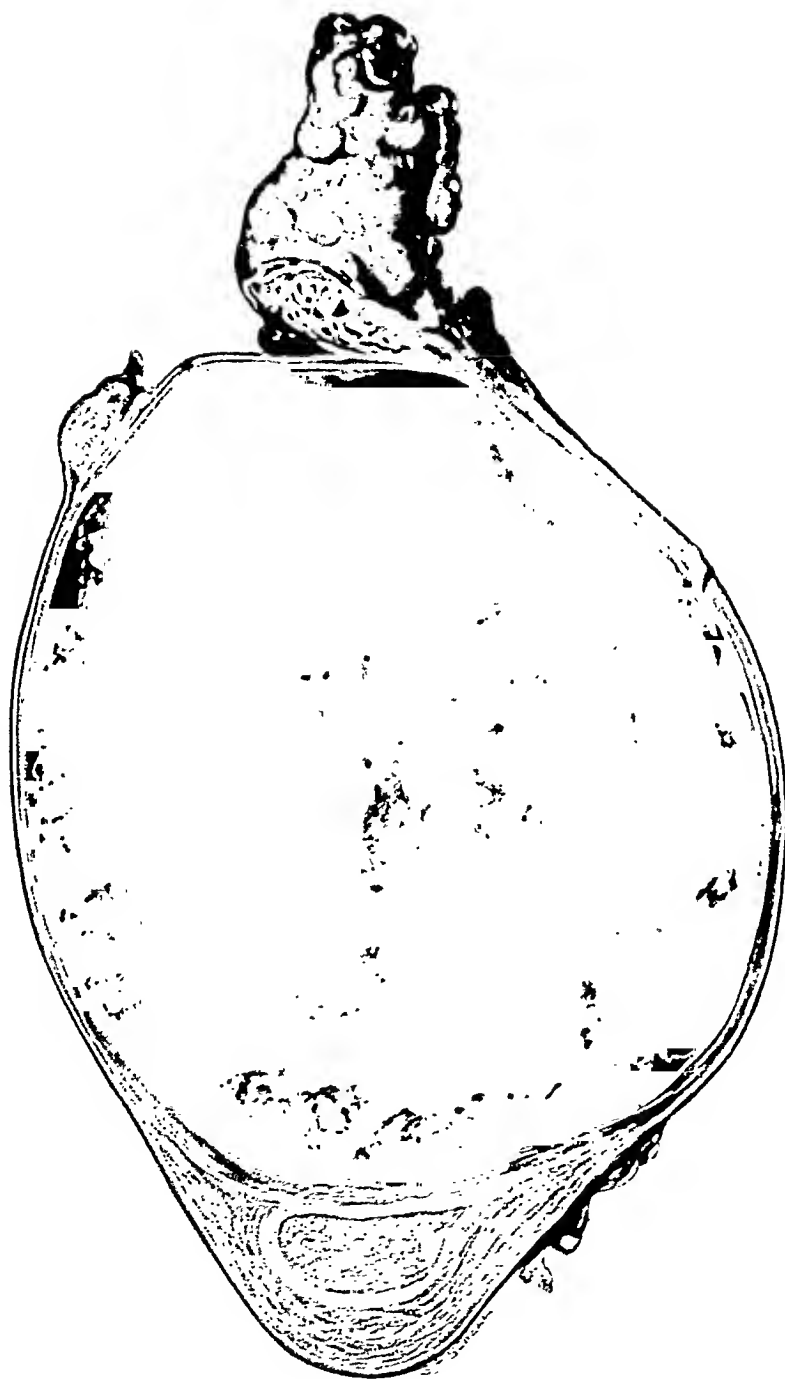
A portion of the spermatic cord, tunica vaginalis, and testis divided by longitudinal section.

The wall of the tunica vaginalis is thickened, as is also the tunica albuginea. Clots, the result of recent hæmorrhage, are attached to the wall of the tunica vaginalis. The testis is shrunken.

Museum of King's College Hospital, 0157A

CLINICAL HISTORY.—There had been a gradual swelling of the left half of the scrotum for three years. This was tapped one month before admission to hospital and dark fluid was obtained. The consistency of the swelling was at first fluid and later became firm. There was no history of injury.

HEMATOCELE.



MUSEUM OF KING'S COLLEGE HOSPITAL, 0157A

LXII. CYST OF THE EPIDIDYMIS.

CYSTS, either single or multiple, are common in the upper pole of the epididymis. They grow slowly and at first spread upwards along the cord. When large they may partially envelop the testis, but the latter always remains separable from the cyst.

A cyst of the epididymis contains a fluid which is either clear or opalescent from the presence of spermatozoa (Spermatocele).

CYST OF EPIDIDYMIS.

(SUPPURATION.)

A right testis, epididymis, and spermatic cord divided by vertical section.

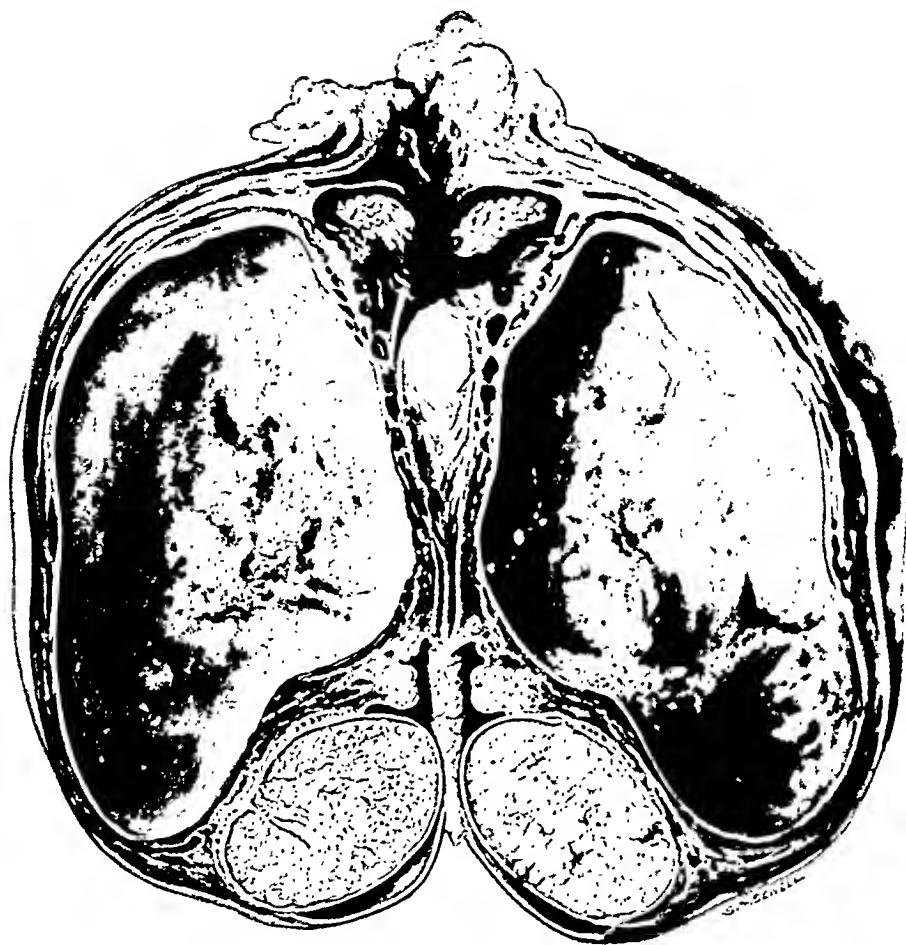
The body of the testis and the tunica vaginalis are normal. There is an oval cyst in the position of the body of the epididymis. Its long diameter measures $3\frac{3}{4}$ in., and it has a thick fibrous wall with a smooth inner lining covered by a thin layer of inflammatory fibrinous exudate. All parts of the specimen are intensely congested, and there are numerous extravasations of blood in the substance of the cyst wall. The body of the testis is situated in front of the lower end of the cyst.

Hunterian Museum, R.C.S., 7983.1

No clinical history.

CYST OF EPIDIDYMISS.

(SUPPURATION.)



HUNTERIAN MUSEUM, R.C.S., 7983.1

CYSTS OF EPIDIDYMIS.

(INFECTION.)

A multilocular cystic tumour divided by longitudinal section.

In the lower part of the specimen is the testis, and in front of this is the moderately distended tunica vaginalis. Above are four cysts of the epididymis. They are lined by exudate as a result of recent infection.

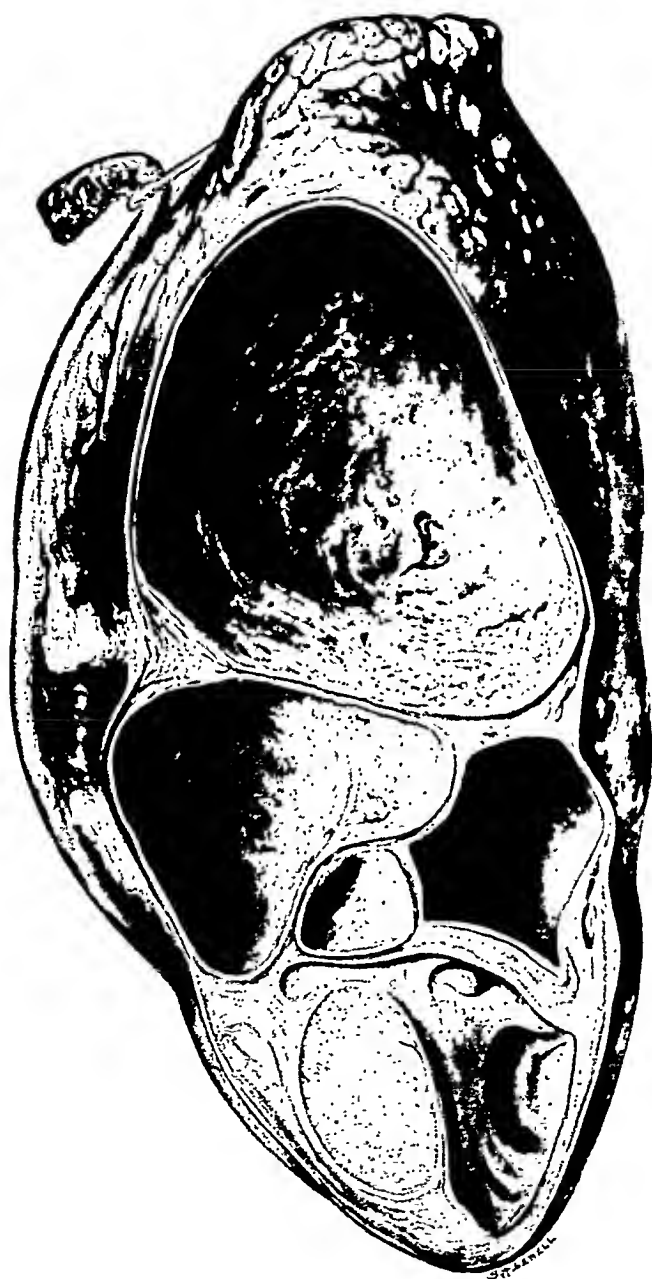
Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 443/14

CLINICAL HISTORY.—The patient was a man, aged 56, who came to hospital complaining of an inflamed swelling in the left side of the scrotum. He said that he had had a hydrocele tapped on this side many times. After the last tapping five days before admission, the scrotum swelled rapidly and its skin became red.

The specimen was removed by operation.

CYSTS OF EPIDIDYMISS.

(INFECTION.)



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE, 443/14

LXIII. TUMOURS OF THE TESTIS.

TUMOURS of the testis show great variety of structure, both to the naked eye and on microscopic examination, but have one feature in common in that they are practically always clinically malignant. Simple tumours of the testis are extremely rare.

It is probable that all the malignant tumours of the testis arise as teratomas, and that the various types become differentiated from one another by overgrowth of one or more elements of the original teratoma. They fall naturally into three main groups: (1) Dermoid cysts; (2) Mixed tumours; (3) Carcinoma and sarcoma of embryonic type.

1. **Dermoid Cyst.**—This is a rare, encapsuled tumour containing a rudimentary embryo, which is usually attached to the wall of a cyst lined by squamous epithelium and containing sebaceous material and hairs. The embryonic portion is often cystic, and may contain representatives of any of the normal tissues of the body.

Clinically, a dermoid of the testis may be recognized as a tumour at any time from birth onwards. It grows slowly and seldom shows any tendency to malignant change.

2. **Mixed Tumour.**—This is a solid or multilocular cystic growth, often of large size. It arises from the rete as an encapsuled tumour, the testis being pushed aside or stretched over it.

Microscopically, the mixed tumour consists of several kinds of embryonic tissue—fibrous tissue, cartilage, muscle, etc. The cysts have a fibrous wall, and are lined by squamous or columnar epithelium from which a carcinoma may develop. Any of these elements may give rise to metastases.

3. **Carcinoma and Sarcoma of Embryonic Type.**—This is a common malignant tumour of the testis, and appears clinically as a soft, vascular growth which rapidly infiltrates and replaces the testis. It has a strong tendency to recur after removal. The recurrences may be local, in the regional lymph-glands, or in distant parts.

Microscopically, the tumour consists of masses of large, spheroidal cells lying in a scanty stroma. The spheroidal cells may show an alveolar arrangement, and the stroma may contain a variable amount of lymphoid tissue.

By many observers this tumour is thought to be distinct from the teratoma and to arise by proliferation of the cells of the seminiferous tubules (Seminoma).

Chorionic carcinoma occasionally arises in a teratoma of the testis, and the original tumour may be so small as to be overlooked until the typical syncytium is recognized in sections of the metastases. It appears to the naked eye both in the testis and in the metastases as a plum-coloured area of hæmorrhage.

TERATOMA OF TESTIS.

(DERMOID CYST.)



A testicle opened from the front.

The testis is enlarged so as to measure $2\frac{1}{2}$ in. in its chief diameter by the growth of a dermoid cyst. An irregularly lobulated process projects into the cyst and carries a tooth and some black hair on its surface.

At the upper part of the section the tunica albuginea is traceable for some distance over the wall of the cyst, and at the back the tunica vaginalis has been reflected to expose the flattened epididymis.

Hunterian Museum, R.C.S., 1233.1

MICROSCOPIC STRUCTURE.—A thin layer of well-developed testicular substance lies stretched out over the wall of the cyst. The interior of the cyst is lined with granulation tissue.

CLINICAL HISTORY.—The patient was a blind boy, aged 16 years, who had suffered from frequent attacks of inflammation in the scrotum after minor injuries.

On examination the right testis was small but situated in the normal position. The left testis was enlarged to form a globular mass in which no distinction between body and epididymis could be found. The skin over it was red and there was a sinus at the junction of scrotum and penis. The testis and overlying skin were removed by operation.

MIXED TUMOUR OF TESTIS.

A testis and epididymis divided by longitudinal section, together with a portion of the spermatic cord.

The body of the testis is completely destroyed by a new growth, the upper part of which is friable and of a pale yellow colour, dotted by hæmorrhages and points of necrosis. The lower part is formed of islets of cartilage, separated by fine connective-tissue strands, with a small quantity of pale-yellow growth. The tumour is encapsuled except at the lower pole, where it is extending into the epididymis. There is no naked-eye evidence that the growth has spread into the cord.

Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 443/10

MICROSCOPIC STRUCTURE.—The tumour is composed of cartilage, myxomatous tissue, and squamous epithelium. Some areas have the histological structure of a large, round-celled sarcoma, such as is commonly seen in the testicle.

CLINICAL HISTORY.—The patient was a man aged 31, who had had a tumour in the testis for one year. During the first ten months it was painless, and it had varied in size from time to time. On examination the testis was enlarged and obscured the outlines of the epididymis. There was no hydrocele. The spermatic cord was not altered. The aortic glands were not enlarged. No abnormality was found on examination of the rectum.

The tumour was removed locally and the immediate recovery was uneventful. After leaving the hospital the patient was treated by Coley's fluid. Eighteen months later he was in good health and free from recurrence.

MIXED TUMOUR OF TESTIS.



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE, 443/10

CARCINOMA OF TESTIS.

One half of a testis with the spermatic cord.

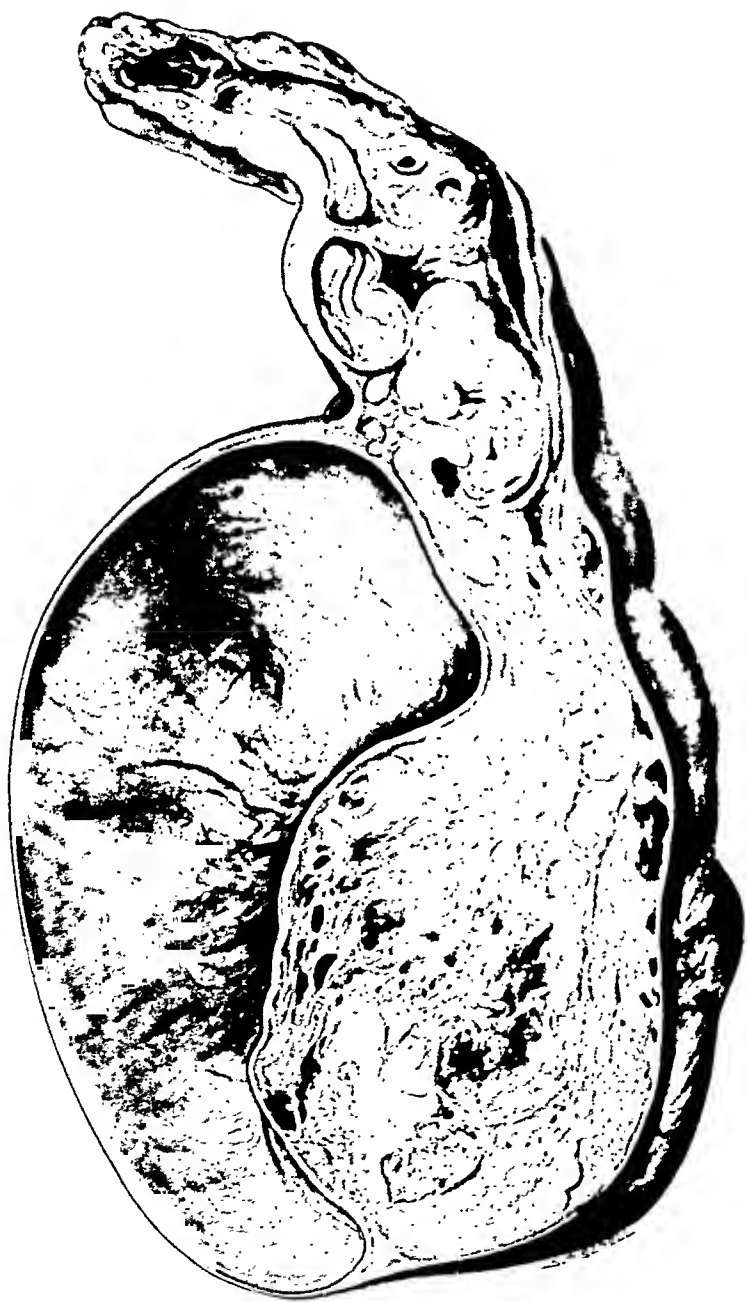
The body of the testis and the epididymis are infiltrated and replaced by a growth which measures $3\frac{1}{2}$ by 2 in. The tumour is firm, and the cut surface shows much fibrous tissue. It extends into the cord for about 2 in., and has distended a vessel which is divided in the section. There is a hydrocele of the tunica vaginalis, and the inner surface of the tunica where it is in contact with the growth is infiltrated and nodular.

Hunterian Museum, R.C.S., 8047.1

MICROSCOPIC STRUCTURE.—Spheroidal-celled carcinoma. In some places the cell masses are large and the stroma scanty; in others the stroma is excessive and the alveoli are slit-like.

No clinical history.

CARCINOMA OF TESTIS.



HUNTERIAN MUSEUM, R.C.S., 8047.1

LXIV. TORSION OF THE TESTIS.

TORSION is more common in the imperfectly descended than in the apparently normal testis, and is probably associated with errors of development in most cases. The immediate cause of the torsion can seldom be determined.

On examination of a recent specimen the spermatic cord is twisted about its long axis immediately above the testis. The latter is usually rotated so that the upper pole lies below. The tunica vaginalis contains a small quantity of blood-stained fluid. The testis is purple or black from extravasation of blood throughout its substance.

TORSION OF TESTIS.



The left testis, epididymis, and spermatic cord.

The body of the testis and the epididymis are deep red from congestion and interstitial hæmorrhage. The epididymis is swollen. There is a thin layer of recent coagulum adhering to the surface of the tunica vaginalis.

Hunterian Museum, R.C.S., 217.2

CLINICAL HISTORY.—The patient was a healthy boy, aged 13 years, who was not known to have had anything wrong with either testis before. One morning he did some jumping, and that evening felt severe pain in the left testis. Two days later there was swelling of the left half of the scrotum. On examination the scrotum was œdematous and the left testis and cord were enlarged and tender. On the fourth day after onset the pain was less but the whole scrotum was red and œdematous.

The parts shown were removed by operation, at which the cord was found to have two complete twists immediately above the testis. The latter was inverted so that the globus major of the epididymis lay below.

LXV. TUMOURS OF THE TESTIS—*continued.*

MIXED TUMOUR OF TESTIS.

A right testis divided by longitudinal section, with 5 in. of the spermatic cord.

In the testis there has grown an oval, polycystic tumour 3 in. in chief diameter. Between the cysts there is a certain amount of connective tissue in which a few small nodules of hyaline cartilage may be seen. The growth is well circumscribed, and has displaced the testicular substance in front of and above it.

With the specimen is preserved half of a polycystic tumour which grew as a metastasis in a lymphatic gland at the root of the left side of the neck and was removed six months after the testis.

Hunterian Museum, R.C.S., 2030.1

MICROSCOPIC STRUCTURE.—Tubules lined with columnar or stratified epithelium lying in a richly cellular connective tissue presenting here and there cylindrical masses of hyaline cartilage.

CLINICAL HISTORY.—The patient was a man, aged 31. An enlargement of the right testis was noticed in February, 1909. The organ was removed in September of the same year. With the testis and spermatic cord one of the lumbar glands was excised. This was the seat of secondary cystic disease. The left testis was retained in the inguinal canal.

Six months after the operation the patient returned with a swelling under the sternal end of the left sternomastoid muscle. This was removed, and proved to be a lymphatic gland. Like the other neoplasms it was cystic, the cysts being lined with epithelium corresponding with that of the spaces in the primary tumour of the testis. The gland had apparently been infected by way of the thoracic duct.

AFTER-HISTORY.—Two years later the patient was in good health.

(*J. Bland Sutton, Lancet, 1909, ii, 1406.*)

MIXED TUMOUR OF TESTIS.



HUNTERIAN MUSEUM, R.C.S., 2650.1

CARCINOMA OF TESTIS.

A testis, tunica vaginalis, and portion of the spermatic cord, divided by longitudinal section.

The testis has been destroyed and replaced by an encapsulated tumour which has extended along the spermatic cord. Its cut surface is irregular, with dense white fibrous areas alternating with areas which are softer and more vascular. There is some interstitial hæmorrhage. The hydrocele contained 3 oz. of clear greenish fluid.

Museum of King's College Hospital, 6210A

MICROSCOPIC STRUCTURE.—Spheroidal-cell carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 45, who was otherwise healthy. For four years he had had an increasing, painless swelling which extended from the position of the right testis into the inguinal canal.

On examination the tumour was stony-hard in some places and fluctuating elsewhere. The tumour ended abruptly at the internal abdominal ring. The overlying skin was normal. There were no palpable glands in the abdomen or groin. A testicular sensation was absent.

The patient also had an epithelioma of the glans penis which was apparently unconnected with the testicular tumour.

CARCINOMA OF TESTIS.



MUSEUM OF
KING'S COLLEGE HOSPITAL,
0210A

MIXED TUMOUR OF TESTIS.

The testis, together with a tumour in its vicinity. The tumour has been divided.

The testis is situated in the upper right corner of the specimen. The tumour consists of three lobes, two of which are lipomatous and one myxomatous in structure.

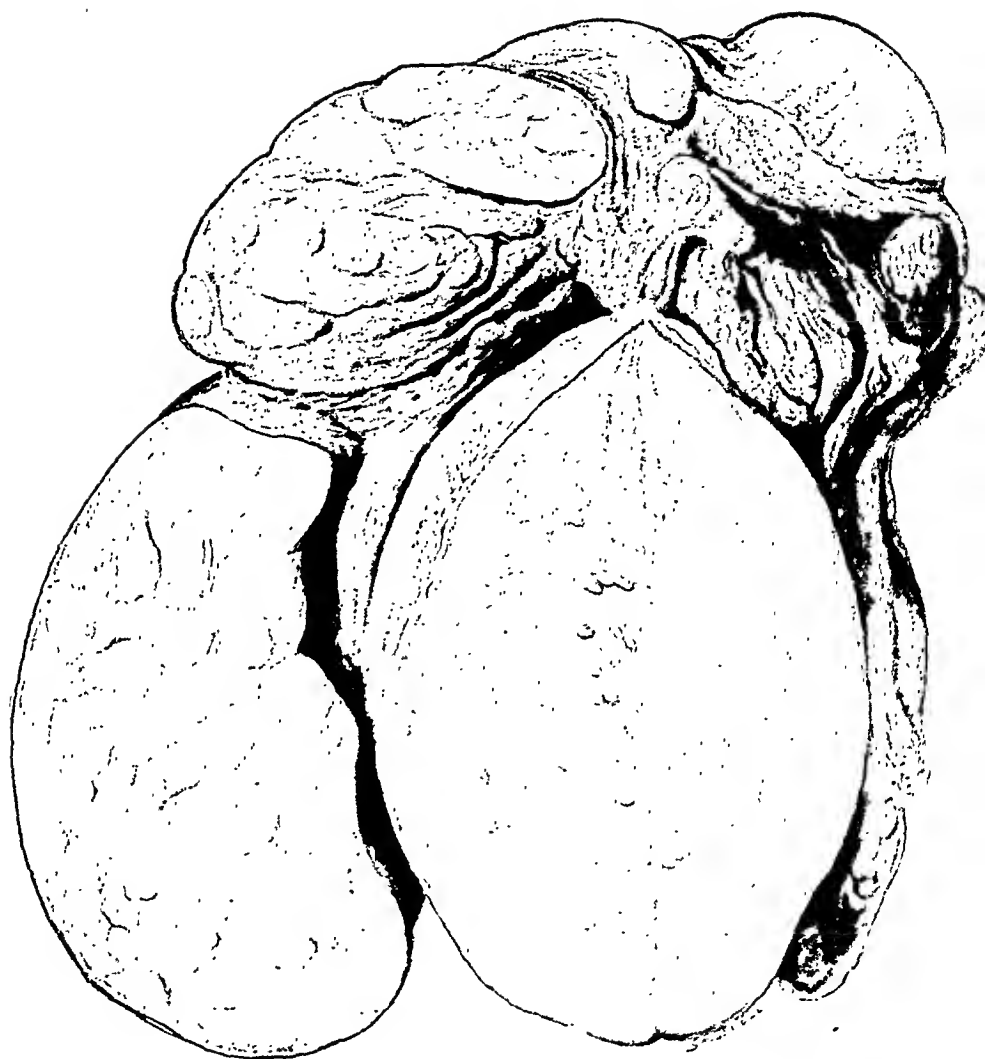
Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 443/15

MICROSCOPIC STRUCTURE.—The two similar portions of the tumour have the structure of a fibro-lipoma. In one of them the cells in the fibrous part are large and their nuclei are hyperchromatic, suggesting cellular activity; there is early myxomatous degeneration. The third portion is a myxomatous fibroma in which there are islands of fat. Although there is no anatomical evidence that the tumour is malignant, some of its characters are suggestive of potential malignancy.

CLINICAL HISTORY.—The patient was a man, aged 72, who had noticed a gradually increasing tumour of the scrotum for six weeks. The size of the swelling was its only inconvenience.

On examination the testis could be palpated in front of the mass. The tumour was removed by operation, and the patient made an uneventful recovery. Twenty months later he was well.

MIXED TUMOUR OF TESTIS.



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE, 442/15

FIBROMYOMA OF SPERMATIC CORD.

A testis divided by longitudinal section together with the spermatic cord.

The testis is normal in structure. Above it is a large, slightly lobulated tumour in connection with the spermatic cord. The cut surface of the tumour is marked by intersecting whorls of fibrous tissue.

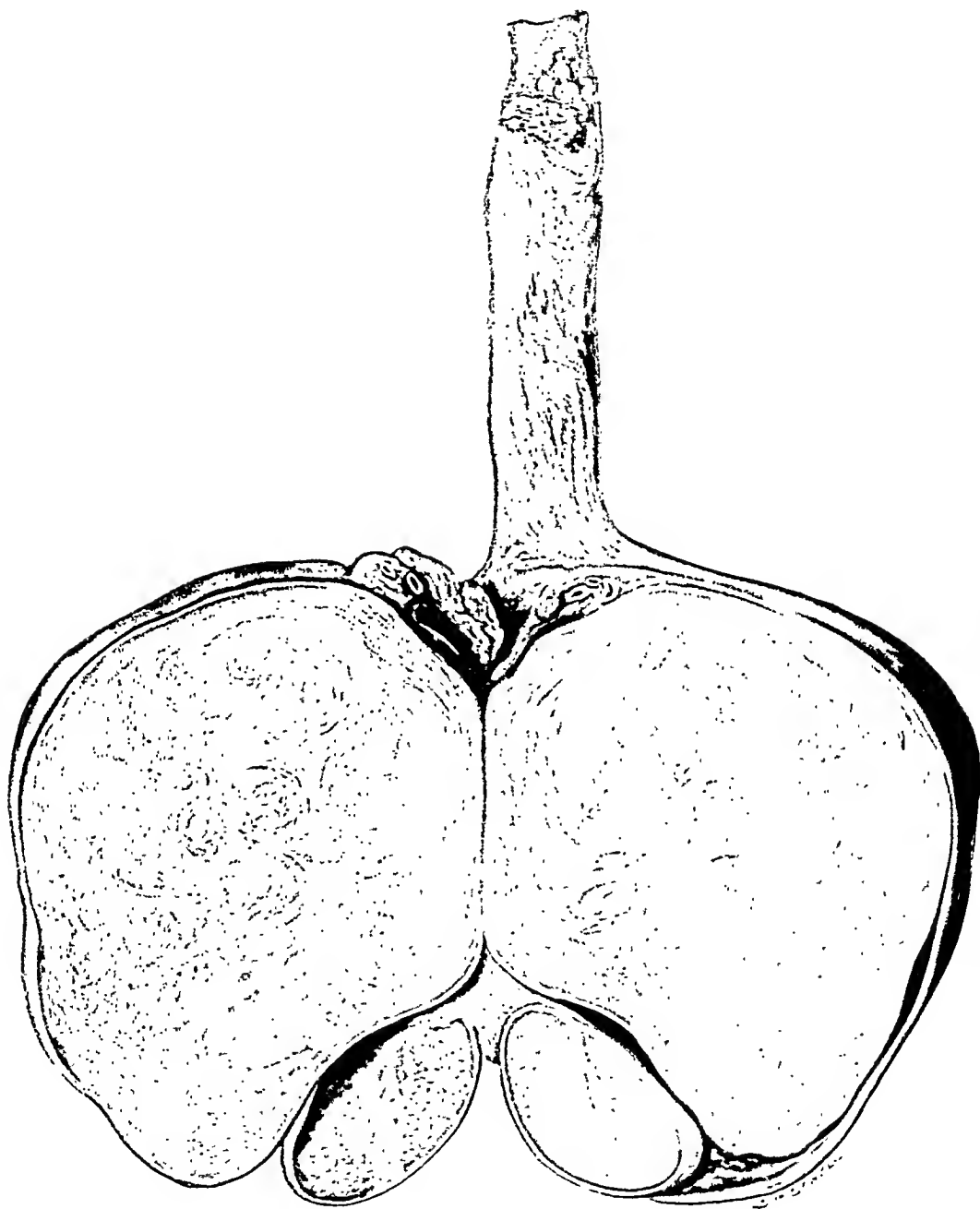
Museum of St. Peter's Hospital for Stone, 2649

MICROSCOPIC STRUCTURE.—The tumour consists chiefly of fibrous tissue, but there is a considerable amount of smooth muscle intermingled with it. Several small areas of calcification are present.

CLINICAL HISTORY.—The patient was a man, aged 36, who first noticed a swelling of the left testicle eighteen months before admission to hospital. It increased slowly in size and occasionally caused a dragging feeling of discomfort in the left groin, but never any actual pain. He had had no injury to that region, nor venereal disease.

On examination a hard, rounded mass was felt in the left half of the scrotum. The scrotal veins were dilated, but the tumour was not attached to the skin. It was closely attached to the upper pole of the testis, which was displaced to its posterior and medial aspect. There were no palpable glands in the inguinal or epigastric regions, and the opposite testis was normal. A provisional diagnosis of testicular neoplasm was made, and the specimen shown was removed.

FIBROMYOMA OF SPERMATIC CORD.



MUSEUM OF ST. PETER'S HOSPITAL FOR STONE, 2649

LXVI. GUMMA OF THE TESTIS.

GUMMA of the testis is a relatively late manifestation of syphilis and may develop many years after the disease was acquired. It affects the body of the testis primarily and produces varying degrees of fibrosis and gumma formation. The epididymis usually remains unaltered, but may share in the fibrous or gummatus changes, and eventually become indistinguishable from the testis. The vas, cord, seminal vesicles, and prostate are not affected. The tunica vaginalis is thickened and at first contains a moderate quantity of fluid. As the disease progresses the fluid may be absorbed and the cavity of the tunica be gradually obliterated by adhesion of its parietal and visceral layers. The appearance of the testis varies according to the relative proportions of fibrosis and gummatus change. Small gummata may develop at several points simultaneously so as to produce a nodular effect on clinical examination. More commonly there is a single large gumma and the testis becomes enlarged, hard, and smooth. On section the tunica albuginea is thickened, and the testicular substance is replaced by syphilitic granulation tissue, fibrous tissue, and necrotic material.

When a gumma softens and comes towards the surface the skin on the anterior aspect of the scrotum becomes œdematous and adherent to the testis. Eventually it breaks down and a gummatus ulcer is formed. The testis may fungate out through the hole. The disease may be bilateral.

Congenital syphilis may produce a diffuse fibrosis and atrophy of one or both testes. Gummata are rare.

LXVII. TUBERCULOSIS OF THE EPIDIDYMIS AND TESTIS.

THE epididymis is one of the common sites in which tuberculosis produces easily recognizable changes. The tubercle bacillus may reach it either along the lymphatics which accompany the vas, or through the blood-stream; but, even in the cases where a blood-borne infection may reasonably be presumed, the vas affords an easy route by which the seminal vesicles and prostate become involved. Hence tuberculosis of the epididymis and testis is usually associated with tuberculosis of the rest of the genital system. Frequently also it is associated with tuberculosis of the urinary tract. In many cases there is evidence of active or quiescent tuberculosis elsewhere or of contact with tuberculous members of the family.

The disease is rare in childhood but is common throughout adult life, particularly between the ages of 20 and 30. It commences on one side as a rule, but, unless checked at an early stage, tends to become bilateral. The common clinical form is insidious in onset and chronic in course, though an acute tuberculous epididymo-orchitis also occurs.

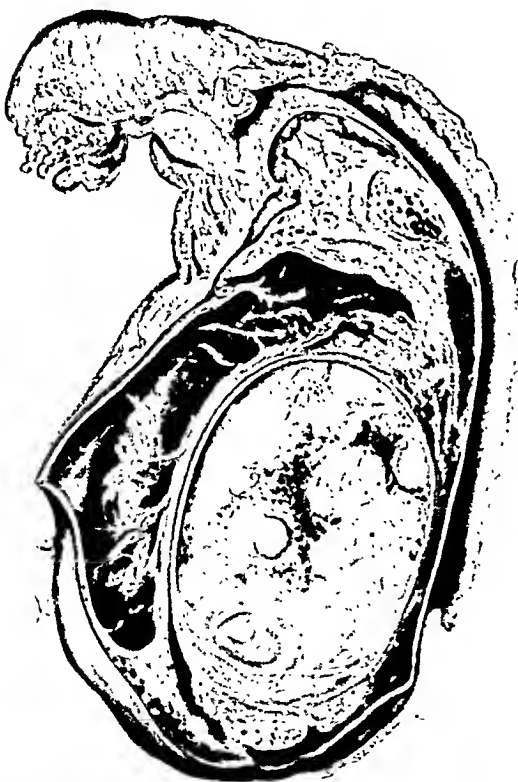
In the chronic variety the lower pole of the epididymis is generally the first site of tuberculous deposit, which appears clinically as a painless, firm nodule. As the disease progresses the remainder of the epididymis becomes involved in a nodular enlargement, the vas becomes thickened either in a uniform or nodular way, and similar nodules may be present in the seminal vesicles and prostate. A small hydrocele often appears in the tunica vaginalis.

When caseation occurs in the tuberculous nodules the overlying skin becomes adherent and œdematous and the liquefied debris tends to point and

to be discharged on the surface. The resulting sinuses are usually situated in the lower and back part of the scrotum, and on the anterior surface where the spermatic cord leaves the upper pole of the testis. The cord as a whole becomes moderately thickened.

The body of the testis is more resistant to tuberculosis than the epididymis and vas, but in late or rapidly developing cases tubercles spread forwards from the epididymis through the mediastinum. If untreated, the tubercles coalesce and caseate, and the testis may eventually be extruded through an ulcerated opening in its coverings. In rare cases, when the balance between the destructive effects of tuberculosis and the resistance of the tissues is sufficiently exact, the scrotal contents become gradually converted into a mass of hard, fibrous tissue in which small areas of caseation may remain.

GUMMA OF TESTIS.



A testis divided by longitudinal section, with part of the spermatic cord.

The cut surface shows that the body of the testis is replaced by a vascular connective tissue in which are several rounded nodules. Two of these are breaking down in the centre.

The epididymis is normal. The tunica vaginalis is distended by a small hydrocele and its cavity is crossed by delicate adhesions.

*Museum of University College
Hospital, 93H2*

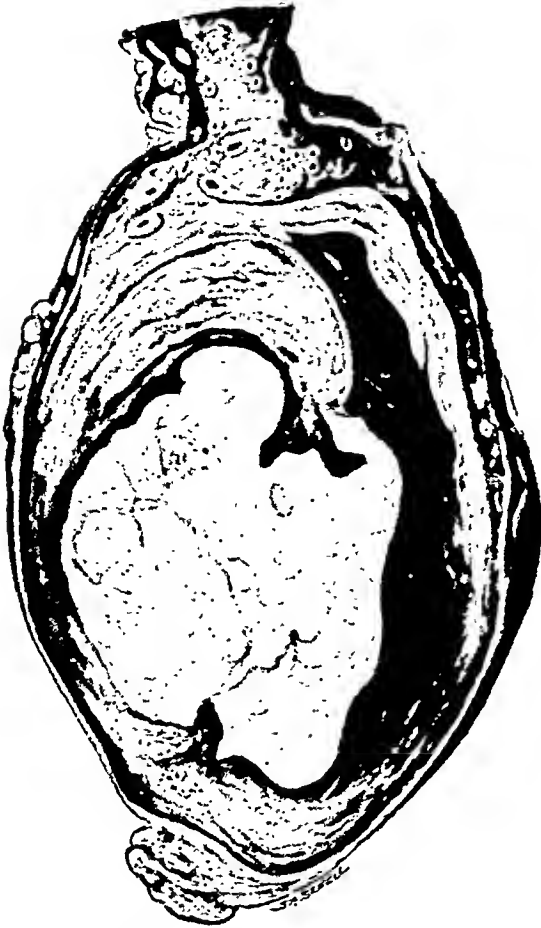
MICROSCOPIC STRUCTURE.— Syphilitic granulation tissue surrounding areas of necrosis. The smaller arteries have greatly thickened walls.

CLINICAL HISTORY.— The patient was a man, aged 58, who had had a gradually increasing swelling of the left testis for nine months. There was continual aching and occasional sharp pain.

On examination the skin of the scrotum was normal; the left testis was enlarged, smooth, and hard; the epididymis was not involved in the swelling; there was no tenderness; the left cord was moderately thickened.

The specimen shown was removed by operation.

GUMMA OF TESTIS.



One half of a testis divided by longitudinal section.

The body of the testis is slightly enlarged, and its central part is occupied by a mass of yellow necrotic tissue with an irregular outline. The necrotic area is separated from the surface of the testis in most parts by a narrow zone of fibrous tissue, but towards the tunica vaginalis it reaches the surface. The cavity of the tunica vaginalis is occupied by blood-clot and exudate, and its wall is greatly thickened. The upper pole of the epididymis is represented by a dense mass of fibrous tissue.

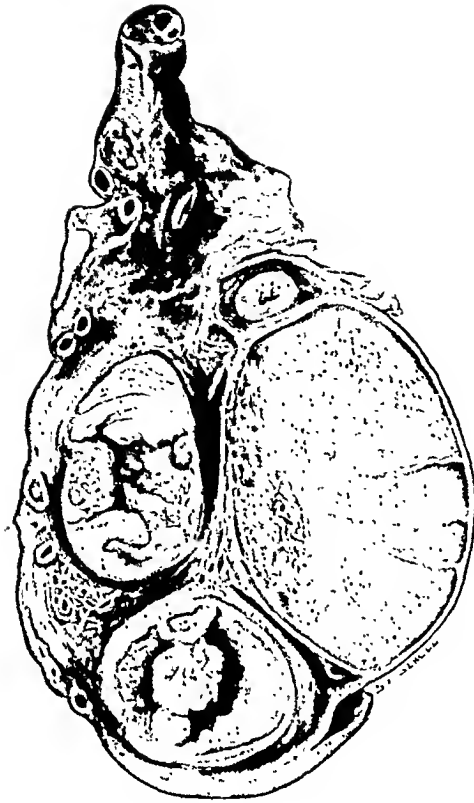
Hunterian Museum, R.C.S., 7962.1

MICROSCOPIC STRUCTURE.—The necrotic area is surrounded by a broad zone of granulation tissue containing numerous fibroblasts but no giant cells.

No clinical history.

TUBERCULOSIS OF EPIDIDYMISS.

A left testis divided by vertical section.



The epididymis is enlarged and contains three cavities filled with caseous material. The body of the testis has many semitranslucent foci scattered throughout it.

Hunterian Museum, R.C.S., 7940.1

MICROSCOPIC STRUCTURE.

Tuberculosis of epididymis. The scattered foci in the body of the testis consist of hyaline connective tissue derived from hyaline degeneration of the tubules and vessels of the gland.

CLINICAL HISTORY.—The patient was a man, aged 30, whose left testicle began to increase in size and to feel tight, though without pain, three weeks before admission to hospital. Twelve years before he had had a stricture following gonorrhœa. An internal urethrotomy was followed by an abscess of the left testicle, which

was opened. Since that time he had had no more urinary symptoms. He did not cough.

On examination he was pale and ill-nourished. The left epididymis was twice the normal size, and at its lower pole was adherent to the skin. The body of the testis felt normal. The vas and surrounding lymphatics were enlarged. There was a hard nodule in the lower pole of the right epididymis. The prostate was enlarged, regular in outline, and free from nodules. The left seminal vesicle was enlarged. The urine contained no tubercle bacilli.

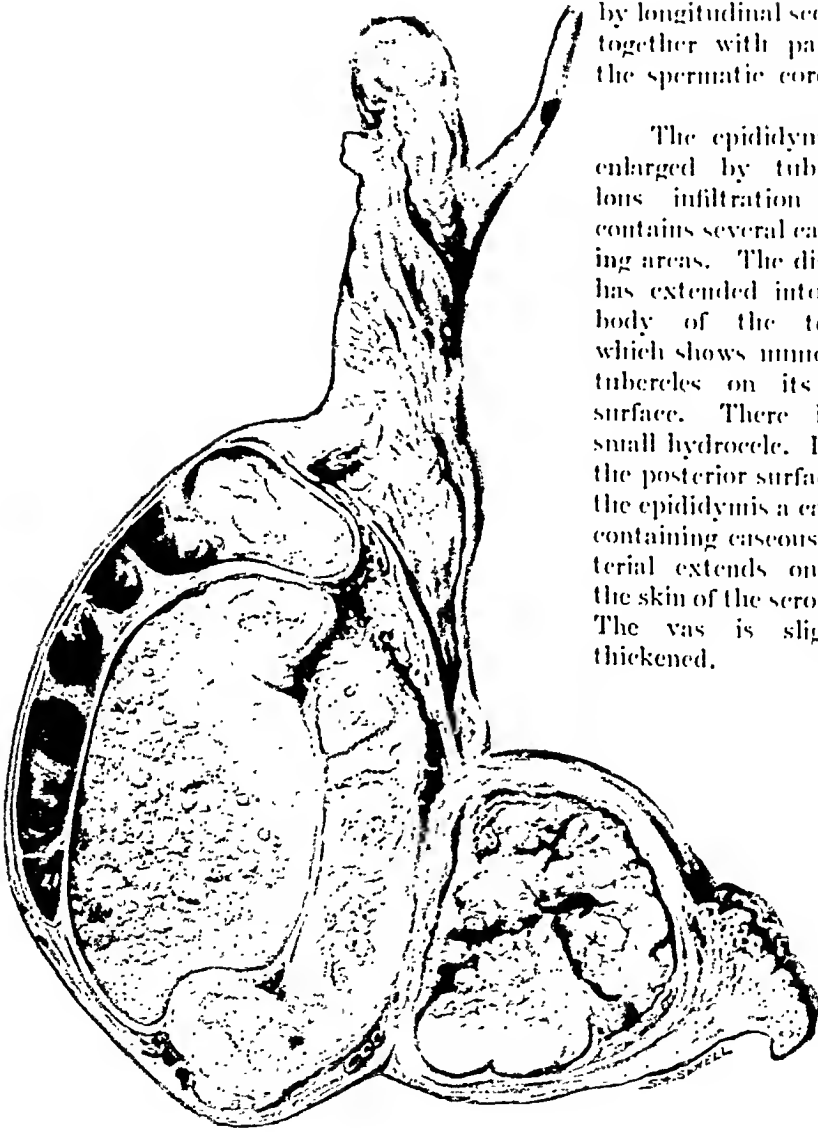
At operation the left testis with 3 in. of the cord was removed. Microscopic examination of the vas showed no disease at the level of the external abdominal ring.

AFTER-HISTORY.—The nodule in the right epididymis disappeared shortly after the operation.

TUBERCULOSIS OF EPIDIDYMS.

A testis divided by longitudinal section, together with part of the spermatic cord.

The epididymis is enlarged by tuberculous infiltration and contains several caseating areas. The disease has extended into the body of the testis, which shows numerous tubercles on its cut surface. There is a small hydrocele. From the posterior surface of the epididymis a cavity containing caseous material extends out to the skin of the scrotum. The vas is slightly thickened.



Museum of St. Peter's Hospital for Stone, 3808

MICROSCOPIC STRUCTURE.—Tuberculosis.

CLINICAL HISTORY.—The patient was a man, aged 33, who had had frequency of micturition and repeated attacks of hæmaturia for one year. The testis had been swollen for four months but was painless and was not tender. The right kidney had been removed for an ulcerating tuberculous lesion three weeks before the orchidectomy.

LXVIII. CARCINOMA OF THE PENIS.

CARCINOMA of the penis is a disease of later life, and is most often seen in patients who suffer from phimosis. It is unknown in circumcised Jews and rare in Mohammedans who practice ritual circumcision at the age of 10 to 12 years. Other predisposing causes are syphilis, leukoplakia, and papillomata of the glans.

It first appears as a warty growth on the glans or at the junction with the prepuce, and usually spreads over the glans and then back along the corpora cavernosa. It seldom causes obstruction of the urethra, but if the urethra is invaded a fistula may develop.

The microscopic structure is usually that of a squamous carcinoma with cell-nests, but an adenocarcinoma derived from the glands around the corona also occurs.

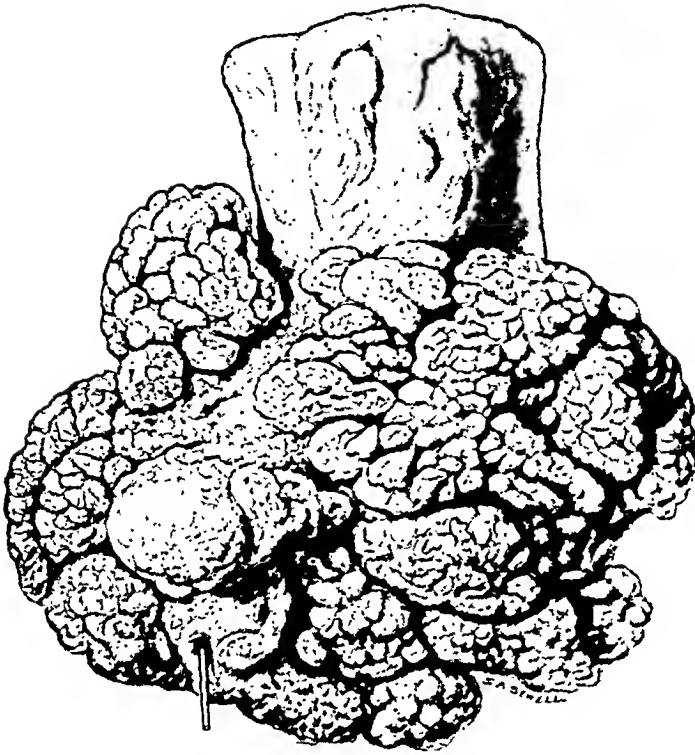
Secondary deposits appear first in the inguinal glands, usually on both sides, and later, when the body of the penis is infiltrated, in the lumbar glands. Metastases elsewhere are rare.

Carcinoma of the penis is locally destructive, but on account of the localization and slow growth of metastases the prognosis is fairly good if adequate removal is possible.

LXIX. CARCINOMA OF THE MALE URETHRA.

CARCINOMA of the male urethra is a rare disease which affects chiefly the bulbous urethra, less commonly the penile, and rarely the prostatic urethra. In about half the cases it supervenes upon a pre-existing stricture. It causes obstruction of the urethra and ulcerates early, giving rise to a purulent or blood-stained discharge. Metastases occur in the external iliac glands, in the glands of the groin, and in distant viscera.

PAPILLOMA OF PENIS.



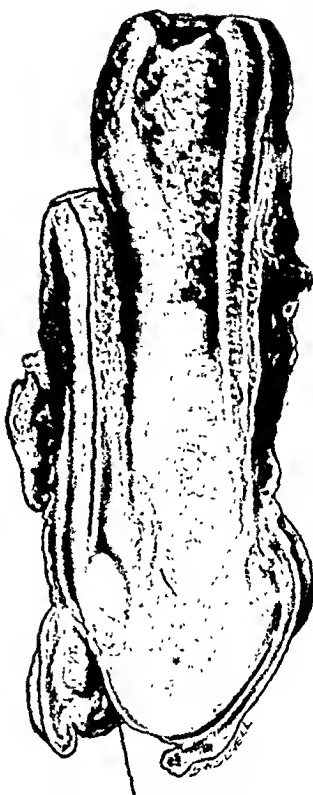
The anterior part of a penis removed after death.

A large lobulated papilloma has grown from the mucous membrane of the glans. The external meatus is completely involved in the tumour and the urethra has been opened behind it, the position of the opening being indicated by a rod.

Hunterian Museum, R.C.S., 1496.1

MICROSCOPIC STRUCTURE.—Benign papilloma.

CLINICAL HISTORY.—The patient was a man, aged 39, who died from an acute visceral disease not connected with that shown in the specimen. The papillomata on the penis had been present for about two years, and were attributed by the patient to venereal disease.



The anterior part of a penis divided by longitudinal section.

The glands in each of the corpora cavernosa are infiltrated by a growth which appears to have originated in the region of the frenum. There is no external lesion elsewhere.

Hunterian Museum, R.C.S., 1898.2

MICROSCOPIC STRUCTURE.—Squamous-celled carcinoma.

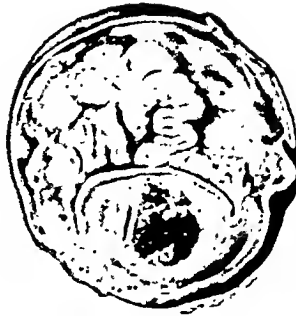
CLINICAL HISTORY.—The patient was a man, aged 64, who dated his symptoms from an injury received three years before; while riding a horse he was jerked up in the saddle and fell forwards, his penis being caught between the saddle and his body. The inguinal and external iliac glands on both sides were indurated.

A complete amputation was performed, but the patient would not consent to an operation on the lymphatic glands.

CARCINOMA OF PENIS.



SURFACE.



SECTION.

A glans penis from the front.

The surface of the glans is covered by an irregular, ulcerated growth which surrounds the margins of the external meatus.

The other illustration shows a cross-section of the penis behind the glans. The carcinoma has extended backwards in the corpora cavernosa.

Museum of St. Peter's Hospital for Stone, 2500

MICROSCOPIC STRUCTURE.—Squamous epithelioma.

CLINICAL HISTORY.—The patient was a man, aged 60, who had noticed irritation at the external meatus for six weeks before admission to hospital. There was some discharge with a little bleeding. He had had difficulty in passing urine for one year. There was no history of venereal disease.

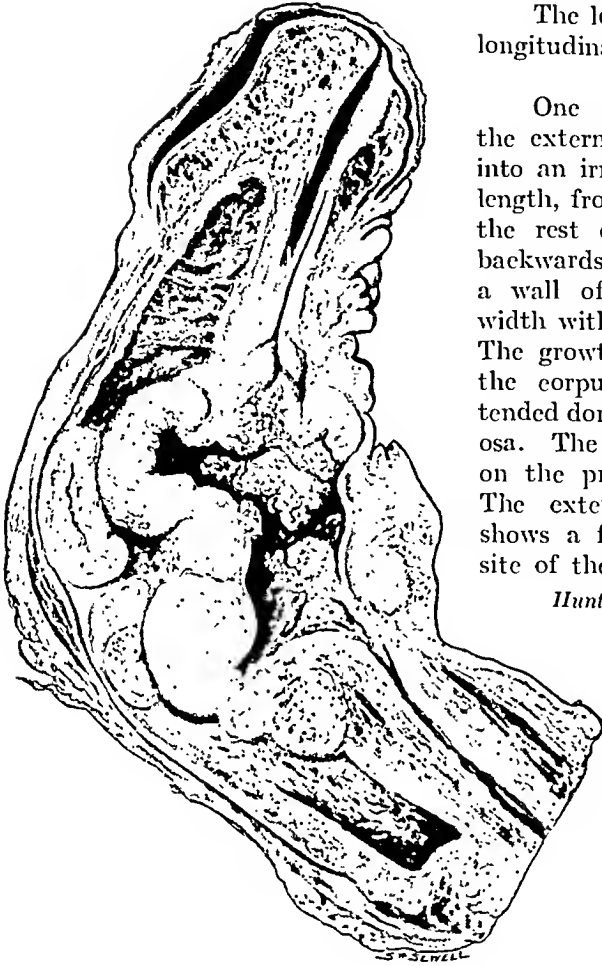
On examination there was an ulcerated growth of the glans; the prepuce was adherent to the glans but was not affected by the neoplasm. There were enlarged glands in both groins and iliac fossæ.

The penis was removed by partial amputation.

No after-history.

CARCINOMA OF URETHRA.

The left half of a penis divided by longitudinal section.



One and three-quarter in. from the external meatus the urethra opens into an irregular cavity about 1 in. in length, from the hinder part of which the rest of the urethra is continued backwards. The cavity is bounded by a wall of white growth of varying width with a nodular external surface. The growth has completely destroyed the corpus spongiosum and has extended dorsally into the corpora cavernosa. The urethra is of normal calibre on the proximal side of the growth. The external surface of the penis shows a fusiform enlargement at the site of the tumour.

Hunterian Museum, R.C.S., 1804.2

MICROSCOPIC STRUCTURE.—A squamous-celled carcinoma consisting mostly of masses of polyhedral cells. There are very few epithelial pearls.

CLINICAL HISTORY.—The patient was a man, aged 59, who had had gonorrhœa at the age of 21. At the age of 53 he

began to have difficulty in passing urine owing to a stricture of the urethra, and for five years was treated by the passage of bougies. About twelve months before his operation he noticed a small hard nodule on the left side of the penis at the junction with the scrotum. After the appearance of this lump he felt increasing pain on the passage of instruments, and eventually dilatation became impossible.

The stricture was excised and on microscopic examination the scar tissue was found to be infiltrated with carcinoma.

A month later a large mass had re-formed and micturition became impossible. The penis was amputated, but the inguinal glands were not removed as no enlargement could be detected.

No after-history.

LXX. MUCOCELE OF THE APPENDIX.

IT happens occasionally that as a result of an attack of acute appendicitis which has stopped short of perforation and has subsided spontaneously a stricture is formed in the proximal part of the appendix or at its junction with the cæcum. If the original infection then dies out or remains of so low an intensity as to stimulate the mucus-secreting cells without leading to further attacks of acute appendicitis, the appendix becomes distended with mucus, and as the pressure within the now closed viscus rises, hernial protrusions of the mucous membrane project through the muscular coat and form diverticula which are recognizable as spherical swellings beneath the peritoneum. Such distension of the appendix may increase slowly until the organ reaches many times its normal size, or at any point its further progress may be cut short by rupture of one of the diverticula. If this occurs the mucus is discharged into the peritoneal cavity, the appendix collapses, the rupture in the diverticulum heals, and the process commences again. This sequence may be repeated at periodic intervals for many years before it causes symptoms or physical signs sufficient to achieve clinical recognition. The extruded mucus is not absorbed by the peritoneum, but becomes encapsulated in the form of droplets or larger masses by omentum or by the growth of filmy connective tissue produced by a local plastic peritonitis. In the course of time the abdomen becomes distended by gelatinous masses (*pseudo-myxoma peritonei*) as in the more common, allied condition caused by rupture of a mucus-secreting ovarian cyst. The production of mucus within the peritoneal cavity in a case of *pseudo-myxoma peritonei* of appendicular origin is usually stopped by removal of the appendix, but in a few cases appendicectomy has had no influence on the progress of the disease. The explanation of this apparent anomaly lies in the occasional escape of mucus-secreting cells from the lining of the appendix during one of its periodic ruptures. The omentum affords a favourable medium upon which such cells may become grafted.

If the possible origin of intraperitoneal mucus from the appendix be not recognized, suspicion may be aroused of the presence of a metastatic colloid carcinoma.

MUCOCELE OF APPENDIX.

A portion of the cæcum with the vermiform appendix.

The appendix is distended to a pyriform sac $3\frac{1}{2}$ in. in length by the accumulation of mucin following upon closure of its cæcal aperture.

Hunterian Museum, R.C.S., 6597.1

CLINICAL HISTORY.—The patient was a woman, aged 54, who died of pneumonia in an asylum of which she had been an inmate for three years. During this time she had had no symptoms which could be referred to the appendix.

MUCOCELE OF APPENDIX.



S. J. SEWELL

HUNTERIAN MUSEUM, R.C.S., 6597.1

MUCOCELE OF APPENDIX.

(DIVERTICULA.)

The anterior half of a cæcum with part of the ascending colon and the appendix. A glass rod has been passed through the ilco-cæcal valve.

The lumen of the appendix is slightly increased, especially in its distal two-thirds. Its walls are thickened and rigid as a result of chronic inflammation. Three small diverticula project from its upper aspect. A large diverticulum protrudes upwards into the cæcum from the proximal end of the lumen of the appendix, and two small diverticula project from its anterior aspect. The larger diverticulum, which almost fills the cæcum, is spherical in aspect, with a diameter of 2 in. There is no communication between the lumen of the appendix and that of the cæcum, and the appendix with its diverticula was distended with mucin in the recent state.

Hunterian Museum, R.C.S., 6602.1

MICROSCOPIC STRUCTURE.—The wall of the appendix consists mostly of bands of dense fibrous tissue and is devoid of glandular elements and lymphoid nodules. It is lined by an imperfect layer of flattened epithelium. The wall of the diverticulum which projects into the cæcum is composed of fibrous tissue without muscle, and is covered by normal mucous membrane on its cæcal aspect. Its inner surface is lined by an incomplete layer of flattened epithelium.

CLINICAL HISTORY.—The patient was a single woman, aged 69, who had had severe attacks of abdominal pain for over a year before admission to hospital. On one occasion she passed a quantity of mucus. She had been somewhat constipated. A fortnight before admission an attack of pain which lasted two days was diagnosed as appendicitis. Her last attack began gradually with abdominal pain which became progressively worse. There was no vomiting. Three weeks after the onset nothing abnormal was found on abdominal examination, but on the following day she had an attack of colicky pain with diarrhoea, and a tumour was then felt running from the right flank to the left of the umbilicus. It formed a well-defined elongated mass, moderately tender and slightly mobile. A diagnosis of intussusception was made.

At operation an intussusception extending to the middle of the transverse colon was readily reduced. It had begun at the fundus of the cæcum and the cæcum was invaginated into the colon, the appendix and terminal ileum being drawn up with it. After reduction of the intussusception there was still a tumour at the fundus of the cæcum, and the parts shown in the specimen were therefore resected.

AFTER-HISTORY.—The patient was in good health four months later.

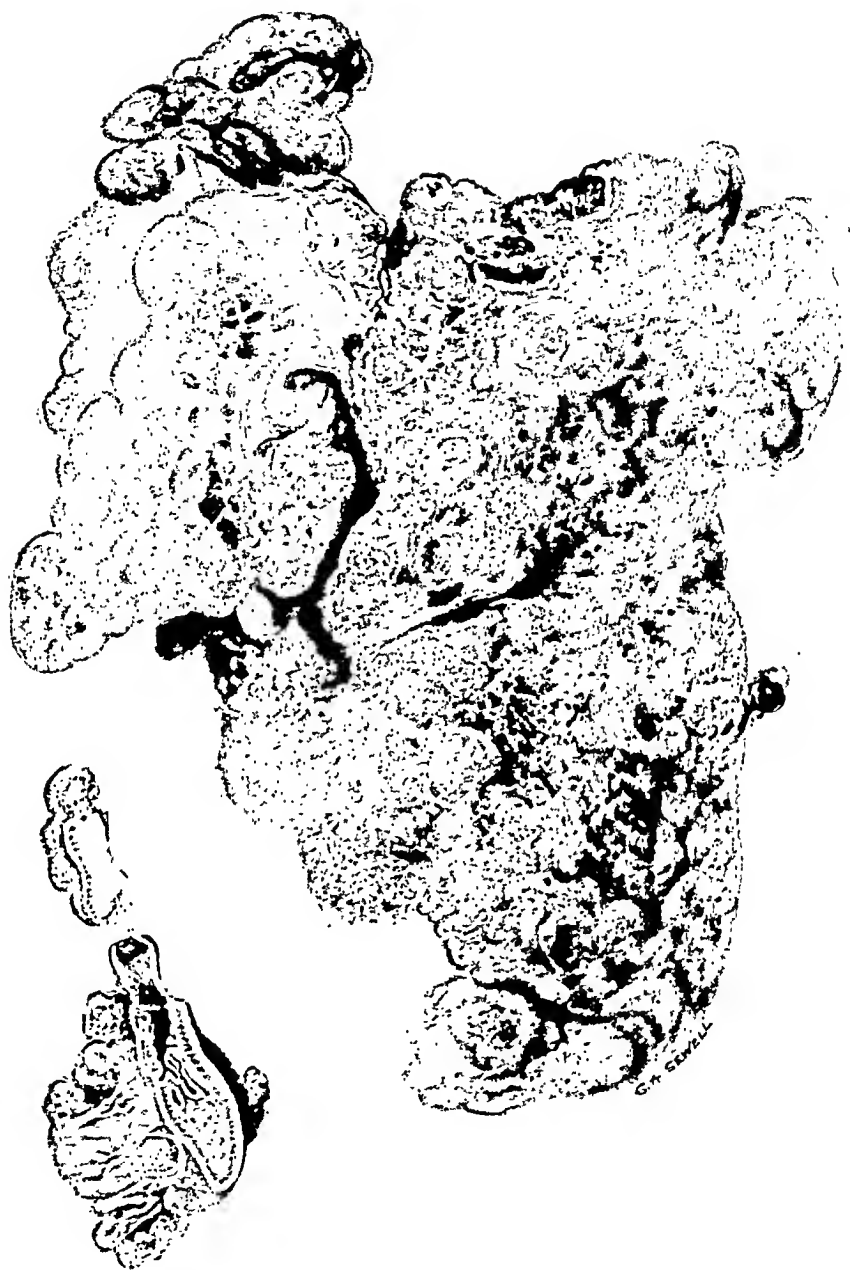
MUCOCELE OF APPENDIX.



—S. S. SEWELL

SURFACE.

HUNTERIAN MUSEUM, R.C.S., 6602.1



HUNTERIAN MUSEUM. R.C.S.. 6599.5 6599.51

LXXI. ACUTE APPENDICITIS.

APPENDICITIS is a very common disease of both sexes throughout the whole of life except in infancy and old age. Its rarity in infancy is probably associated with the simplicity of infant diet, and in old age with progressive atrophy of the appendix.

The micro-organisms involved are those which normally inhabit the large bowel, streptococci and coliform bacilli being most often found.

The chief local factor which can be recognized as predisposing to acute appendicitis is the presence of a stercolith, an inspissated mass of faecal material which the normal peristaltic activity of the appendix is unable to expel into the caecum. In a few cases appendicitis appears to start as part of an extensive infection of the intestinal tract, which subsides except for that part which involves the appendix. In a large proportion of cases no definite cause for the attack can be deduced from a study of the morbid anatomy of the appendix.

All degrees of acute appendicitis are met with, from a mild state of inflammation to gangrene which may involve a part or the whole of the organ. In the early stages of a relatively slight attack the appendix is thickened by inflammatory oedema and exudate into its walls, the peritoneal coat is red and loses its lustre, and flakes of lymph appear on its surface. The meso-appendix and lymph-glands of the lower ileal mesentery share in the inflammation. A fluid exudate containing numerous phagocytes is poured out in varying quantity into the neighbouring part of the peritoneal cavity, and the omentum often becomes wrapped round and adherent to the inflamed appendix.

On microscopic examination the appendix is infiltrated with leucocytes, which are concentrated chiefly in the submucous and subperitoneal coats, where interstitial abscesses may form. The lining epithelium desquamates and leaves small areas of ulceration in the mucous membrane. The vessels show damage by thrombosis and by hæmorrhage into the wall and into the lumen of the appendix. Such interstitial extravasation of blood may lead to a brown staining of the mucous membrane which remains as permanent evidence of a preceding attack of appendicitis. When ulceration of the mucosa is deep or prolonged its healing may be followed by sufficient scarring to produce a stricture, which is then a potent cause of further attacks of appendicitis.

The more severe cases of appendicitis which end in perforation present in their earlier stages two clinical types characterized by the predominance of signs either of inflammation in the right iliac fossa or of obstruction to the lumen of the appendix.

Acute appendicular obstruction may be brought about by the contraction of a stricture resulting from ulceration in a previous attack of appendicitis which has resolved spontaneously, by the presence of a stercolith around which the mucous membrane becomes swollen until the lumen is blocked, or by inflammatory swelling of the mucosa in an appendix which is bent abruptly by a peritoneal fold, whether this occurs naturally or is the result of adhesions following a previous attack of appendicitis.

Perforation may occur at any point in the appendix, and is preceded by the development of a localized patch of gangrene in the wall. It is often situated over a stercolith because of the additional factor of pressure of the swollen mucous membrane on the incompressible stercolith. In such a case the stercolith is often ejected with the pus through the perforation and may escape observation at operation.

In rare cases gangrene of the whole or of the distal part of the appendix follows thrombosis of the main appendicular vessels, and if the resulting peritonitis is localized the appendix with the remains of its mesentery may be found lying free in an abscess cavity or wrapped in a cocoon of omentum.

The more severe grades of peritonitis are usually associated with perforation, but may also follow the passage of micro-organisms through the wall of an inflamed appendix which appears intact to the naked eye. When the original infection is intense and rapid in its development the resulting peritonitis spreads progressively without limitation by inflammatory adhesions. Spreading peritonitis is the usual result of perforation of an obstructed appendix, and may be beyond control within six hours of the first onset of pain. A localized abscess is formed when the development of the appendicitis is slower and time is allowed for the formation of protective adhesions.

Torsion and Intussusception of the Appendix are rare.

ACUTE APPENDICITIS.



A vermiform appendix removed by operation and divided by longitudinal section.

The mucous membrane is swollen and hæmorrhagic. The submucosa is infiltrated by pus except towards the apex, and the lumen is occupied by hæmorrhagic exudate. The surface is congested, and with the adjacent mesentery is covered with a layer of fibrous exudate.

Hunterian Museum R.C.S., 6577.1

No clinical history.

ACUTE APPENDICITIS.

A vermiform appendix.

The appendix and its mesentery are swollen and intensely congestive and in many places are covered by purulent exudate. The terminal part of the appendix is dilated, and one inch from its tip is a perforation from which a faecal concretion protrudes. The appendix is free from fibrous adhesions.

Hunterian Museum R.C.S., 6579.1

CLINICAL HISTORY.—The patient was a girl, aged 19, who had had four subacute attacks of appendicitis. The onset of the last attack was sudden, with rapidly increasing abdominal pain followed after three hours by vomiting, which recurred a few hours later.

On examination the abdomen was rigid and tender, the tongue was coated, and the pulse 126.

At operation ten hours after the onset of the attack the appendix was removed from a retroæcal position.

The patient made a good recovery.

ACUTE APPENDICITIS.



HUNTERIAN MUSEUM, R.C.S., 6579.1

SUBACUTE APPENDICITIS.

Part of a cæcum with a vermiform appendix divided by longitudinal section.

The appendix is greatly enlarged by inflammation, and forms a curved, cylindrical mass $3\frac{1}{2}$ in. by $1\frac{1}{2}$ in. It is covered by smooth, congested peritoneum, beneath which is a thick layer of fibrous tissue. The lumen of the appendix is visible in the upper part of this mass and appears again in transverse section near the apex. The mucous membrane is thickened both in the cæcum and in the appendix. In the fatty tissue beside the cæcum are several slightly swollen lymph-glands.

Hunterian Museum R.C.S., 6595.2

MICROSCOPIC STRUCTURE.—The mucous membrane is thickened by a leucocytic exudate and its lymph-follicles are increased in size. The dense tissue round the appendix consists of hyaline fibrous tissue in which are numerous small foci of leucocytes.

CLINICAL HISTORY.—The patient was a girl, aged 16. Two months before operation she complained of pain in the lower abdomen. This gradually increased and three weeks after the onset was accompanied by a rise of temperature. There was no vomiting.

On examination a tender mass was felt in the right iliac fossa.

At operation the appendix was adherent to the anterior abdominal wall, and was excised with part of the cæcum and the terminal ileum.

The patient made a good recovery.

SUBACUTE APPENDICITIS.



HUNTERIAN MUSEUM, R.C.S., 6595.2



A vermiform appendix divided longitudinally.

The mucous membrane is slightly thickened as a result of inflammation. It is brown in colour.

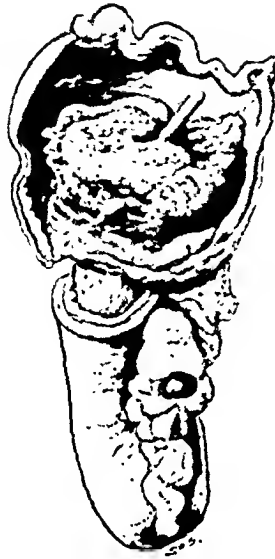
Hunterian Museum, R.C.S., 6604.1

MICROSCOPIC STRUCTURE.—The pigment is in the form of fine, brown granules arranged in broken vertical lines in the connective tissue of the mucous membrane. The granules of pigment are contained in large cells which are arranged in clusters in the mucous membrane and are seen here and there singly in the submucosa. There is no pigment in the muscular coat nor in the lymph-nodes of the mucosa. Microchemical reactions show that it is not derived from ingested iron.

CLINICAL HISTORY.—The patient was a woman, aged 48, who had suffered since the age of 21 from constipation which necessitated the use of purgatives. For the last fourteen years she had had occasional attacks of pain and tenderness in the right iliac fossa, and for three years had been treated intermittently for colitis. In the four months preceding operation there had been four attacks of tenderness in the right iliac fossa, and during one of these there had been a severe rise of temperature.

(*W. H. Battle, Proc. Roy. Soc. Med.*, 1915, viii (Surg. Sect.), 26 ;
and *S. G. Shattock, Proc. Roy. Soc. Med.*, 1916, ix (Path. Sect.), 43.)

INTUSSUSCEPTION OF APPENDIX.



A vermiform appendix with the adjacent part of the caecum.

The proximal half of the appendix has been intussuscepted into itself and then into the caecum, where it forms an irregular, deep purple mass nearly one inch in diameter. The distal part of the appendix is somewhat thickened by inflammation.

A white rod has been inserted into the lumen of the intussuscepted part of the appendix.

Hunterian Museum, R.C.S., 6564.1

CLINICAL HISTORY.—The patient was a boy, aged 6 years, who had suffered from persistent vomiting for six days with some pain in the right iliac fossa but no rigidity or tenderness. He had been constipated. There was no blood in the stools. The parts shown were removed by operation.



A vermiform appendix.

The appendix is greatly distended and measures 4 in. in length by 1 in. in diameter. At its proximal end it is tightly twisted with its mesentery about its long axis. Both appendix and meso-appendix are of a dull purple colour and are partly covered by a fibrinous inflammatory exudate. The superficial veins of the proximal part are tensely distended with blood.

The enlargement of the appendix is to be attributed mainly to recurrent attacks of obstruction due to lesser degrees of torsion.

Hunterian Museum, R.C.S., 6562.1

CLINICAL HISTORY.—The patient was an ex-soldier who had suffered with pain in the right iliac fossa and vomiting for four days. The appendix was removed by operation.

